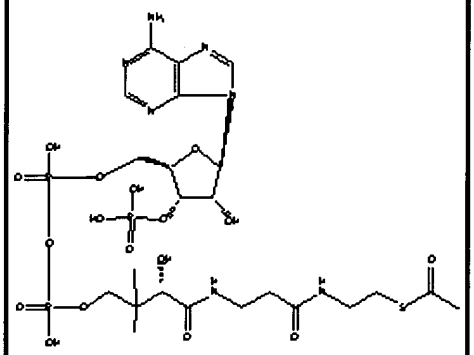
L Number	Hits	Search Text	DB	Time stamp
1	121	(536/26.23).CCLS.	USPAT; US-PGPUB	2003/10/03 17:13
2	332	(514/47).CCLS.	USPAT; US-PGPUB	2003/10/03 17:14
3	424	((536/26.23).CCLS.) ((514/47).CCLS.)	USPAT; US-PGPUB	2003/10/03 17:14
5	46	((536/26.23).CCLS.) AND (fatty OR palm$ or stear$ OR oleo$ OR arachid$)	USPAT; US-PGPUB	2003/10/03 17:15
4	229	(((536/26.23).CCLS.) ((514/47).CCLS.)) AND (fatty OR palm$ or stear$ OR oleo$ OR arachid$)	USPAT; US-PGPUB	2003/10/03 17:16
6	156	(((536/26.23).CCLS.) ((514/47).CCLS.)) AND (fatty OR palm$ or stearoy$ OR oleo$ OR arachid$)	USPAT; US-PGPUB	2003/10/03 17:17
7	148	(((536/26.23).CCLS.) ((514/47).CCLS.)) AND (fatty OR palmit$ or stearoy$ OR oleo$ OR arachid$)	USPAT; US-PGPUB	2003/10/03 17:20
8	24	((536/26.23).CCLS.) AND ((fatty NEAR25 ester) OR palmit$ or stearoy$ OR oleo$ OR arachid$)	USPAT; US-PGPUB	2003/10/03 17:23
9	6	(((536/26.23).CCLS.) ((514/47).CCLS.)) AND (ACETYL adj CoA)	USPAT; US-PGPUB	2003/10/03 17:24

acetyl-CoA [72-89-9]

Synonyms: acetyl-CoA; acetyl coenzyme A;



Tools

BUY AT CHEMACX.COM
VIEW CHEMDRAW STRUCT
VIEW CHEM3D MODEL

CAS RN Lookup

THE MERCK INDEX
NCI DATABASE

OpenChem

VIEW LINKS
ADD COMPOUND
ADD/CHANGE PROPERTY
ADD LINK

Formula	$C_{23}H_{38}N_7O_{17}P_3S$	Molecular Weight	809.57118
CAS RN	72-89-9	Melting Point (°C)	
ACX Number	X1013150-1	Boiling Point (°C)	
Density		Vapor Density	
Refractive Index		Vapor Pressure	
Evaporation Rate		Water Solubility	
Flash Point (°C)		EPA Code	
DOT Number		RTECS	
Comments			

More information about the chemical is available in these categories:

Biochemistry (4)

Biocatalysis/Biodegradation Database

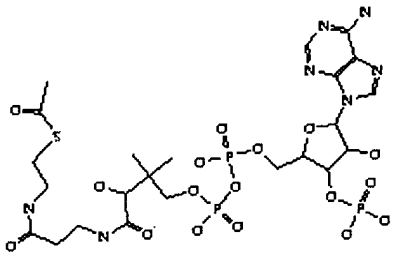
Information about this particular compound

Isoprenoid Pathway

Ligand Chemical Database for Enzyme Reactions

Check ligand chemistry at ChemPDB (EBI)

PDBsum list of PDB entries and other information for "ACO"



PDB list of entries that contain ACO

Entry for "ACO" in the Hetero Components Database (Jena)

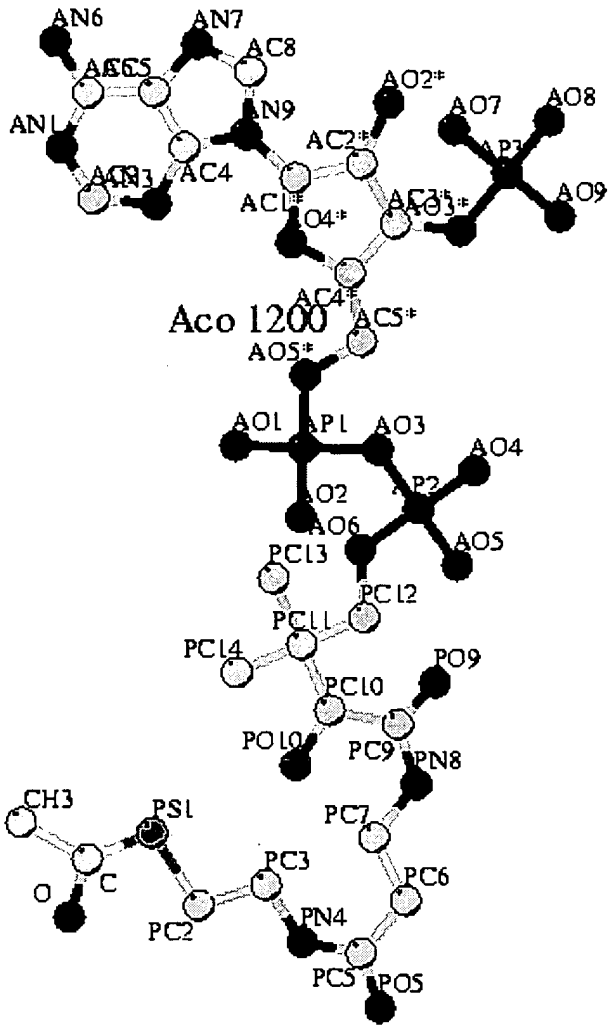
Check Relibase (CCDC) [ACO]

NIST Chemistry Webbook hits for formula C23 H38 N7 O17 P3 S1

Summary of HETZE report :

```
Residue type                    : (ACO)
Identifier                      : (  535)
Segment ID                      : ( )

Nr of atoms                     : (      51)
List of elements (from file)    : ( C23 H38 N7 O17 P3 S1)
Deduced formula                 : (C23 N7 O17 P3 S1)
Guestimated total nr of Hs      : (      43)
Nr of extra examples            : (       0)

Nr of distances < 0.8 A         : (       0)
Nr of bond angles < 80 degrees  : (       0)

Nr of bonds found               : (      53)
... bonds without ideal value   : (       0)
... bonds near ideal value      : (      42)
... bonds far from ideal value  : (      11)
         -"-                  % : (  20.755)

Nr of angles found              : (      79)

Nr of dihedrals found           : (      90)

Nr of atoms with impropers      : (      16)
... imprs far from ideal value  : (       0)
         -"-                  % : (   0.000)
```

Nr of flat planes : (15)



- *This page is part of the HIC-Up site (Hetero-compound Information Centre - Uppsala).*
- *© 1997 - 2003, Gerard Kleywegt, Uppsala, Sweden.*
- *Please read this before contacting Gerard.*
- *Sign or view the HIC-Up guestbook - thanks !*
- *HIC-Up release 7.2 [2003-04-17].*

Created at Fri Apr 18 06:58:38 2003.

000455

HIC-Up

Acronym:	**ACO**
PDB entry:	**1i12 (PDB) (PDBsum) (EDS) (MSD) (MMDB)**
Also in:	**16 other PDB entries, including 1GHE 1HM9 1M3Z 3CSC 4CSC 1DM3 1QSR 1BOB 1QSM 1HM8 1KRR 1MJB 1B87 1KK4 1OD2**
Formula:	**C23 H38 N7 O17 P3 S1**
Resolution (Å):	**1.30**
Name(s):	• **acetyl coenzyme a** • **cobalamin-adenosine complex**

WARNING - alternative chemical formulas found: C23 H38 N7 O17 P3 S1 ... C22 H39 N7 O18 P3 S1 ... C72 H100 N18 O17 CO1 P1

| Coordinates | Visualisation | Dictionaries | Miscellaneous | Off-site | Rest |

Coordinates

PDB file (".pdb"; with REMARK and HETATM records)

PDB file (".txt"; with REMARK and HETATM records)

Clean PDB file (".pdb"; no REMARKs; ATOM records)

Clean PDB file (".txt"; no REMARKs; ATOM records)

Visualisation

VRML file (gzip-ed)

ChemScape Chime page

Dictionaries

PDB dictionary file (CONECT records, etc.)

X-PLOR/CNS topology file

X-PLOR/CNS parameter file (0 warnings, 3 notes)

X-PLOR/CNS energy minimisation input file

O RS_FIT datablock

O RSR datablock

O connectivity entry

O torsion entry

O Refi dictionary entry

TNT dictionary file

Miscellaneous

Disclaimer

HETZE log file (rudimentary quality assessment)

Connection table not generated for this entity

Off-site

EDS density (Java2 applet) for ACO 1200 in PDB entry 1i12

Generate hydrogens and dictionaries (MOL2, GROMOS87, GROMACS, WHAT IF, HEX, CNS, O, and SHELX) using the PRODRG server in Dundee:

Run PRODRG

Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:ssspta1611txm

PASSWORD:
TERMINAL (ENTER 1, 2, 3, OR ?):2

* * * * * * * * * * Welcome to STN International * * * * * * * * * *

NEWS 1		Web Page URLs for STN Seminar Schedule - N. America
NEWS 2		"Ask CAS" for self-help around the clock
NEWS 3	SEP 09	CA/CAplus records now contain indexing from 1907 to the present
NEWS 4	Jul 15	Data from 1960-1976 added to RDISCLOSURE
NEWS 5	Jul 21	Identification of STN records implemented
NEWS 6	Jul 21	Polymer class term count added to REGISTRY
NEWS 7	Jul 22	INPADOC: Basic index (/BI) enhanced; Simultaneous Left and Right Truncation available
NEWS 8	AUG 05	New pricing for EUROPATFULL and PCTFULL effective August 1, 2003
NEWS 9	AUG 13	Field Availability (/FA) field enhanced in BEILSTEIN
NEWS 10	AUG 15	PATDPAFULL: one FREE connect hour, per account, in September 2003
NEWS 11	AUG 15	PCTGEN: one FREE connect hour, per account, in September 2003
NEWS 12	AUG 15	RDISCLOSURE: one FREE connect hour, per account, in September 2003
NEWS 13	AUG 15	TEMA: one FREE connect hour, per account, in September 2003
NEWS 14	AUG 18	Data available for download as a PDF in RDISCLOSURE
NEWS 15	AUG 18	Simultaneous left and right truncation added to PASCAL
NEWS 16	AUG 18	FROSTI and KOSMET enhanced with Simultaneous Left and Righ Truncation
NEWS 17	AUG 18	Simultaneous left and right truncation added to ANABSTR
NEWS 18	SEP 22	DIPPR file reloaded
NEWS 19	SEP 25	INPADOC: Legal Status data to be reloaded
NEWS 20	SEP 29	DISSABS now available on STN

NEWS EXPRESS	OCTOBER 01 CURRENT WINDOWS VERSION IS V6.01a, CURRENT MACINTOSH VERSION IS V6.0b(ENG) AND V6.0Jb(JP), AND CURRENT DISCOVER FILE IS DATED 23 SEPTEMBER 2003
NEWS HOURS	STN Operating Hours Plus Help Desk Availability
NEWS INTER	General Internet Information
NEWS LOGIN	Welcome Banner and News Items
NEWS PHONE	Direct Dial and Telecommunication Network Access to STN
NEWS WWW	CAS World Wide Web Site (general information)

Enter NEWS followed by the item number or name to see news on that specific topic.

All use of STN is subject to the provisions of the STN Customer

agreement. Please note that this agreement limits use to scientific research. Use for software development or design or implementation of commercial gateways or other similar uses is prohibited and may result in loss of user privileges and other penalties.

* * * * * * * * * * * * * * * STN Columbus * * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 10:10:13 ON 04 OCT 2003

=> file reg

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'REGISTRY' ENTERED AT 10:10:33 ON 04 OCT 2003
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

Property values tagged with IC are from the ZIC/VINITI data file provided by InfoChem.

STRUCTURE FILE UPDATES: 1 OCT 2003 HIGHEST RN 596788-60-2
DICTIONARY FILE UPDATES: 1 OCT 2003 HIGHEST RN 596788-60-2

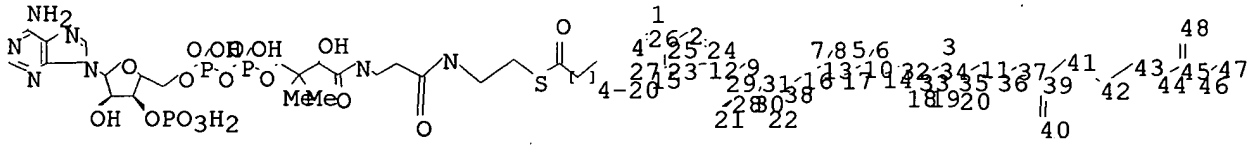
TSCA INFORMATION NOW CURRENT THROUGH JULY 14, 2003

Please note that search-term pricing does apply when conducting SmartSELECT searches.

Crossover limits have been increased. See HELP CROSSOVER for details.

Experimental and calculated property data are now available. See HELP PROPERTIES for more information. See STNote 27, Searching Properties in the CAS Registry File, for complete details:
http://www.cas.org/ONLINE/STN/STNOTES/stnotes27.pdf

=>
Uploading C:\Program Files\Stnexp\Queries\10070439.str



chain nodes :
1 3 5 6 7 8 10 11 13 14 16 17 18 19 20 21 22 32 33 34 35 36 37 38 39 40 41 42 43 44 45 46 47 48
ring nodes :
2 4 9 12 15 23 24 25 26 27 28 29 30 31
chain bonds :
1-26 3-34 5-10 6-10 7-13 8-13 10-17 10-14 11-35 11-36 12-29 13-16 13-17 14-32 16-38 18-33 19-33 20-35 21-28 22-30 31-38 32-33 33-34 34-35 36-37 37-39 39-40 39-41 41-42 42-43 43-44 44-45 45-46 45-48 46-47
ring bonds :
2-24 2-25 4-26 4-27 9-29 9-31 12-23 12-24 15-23 15-27 23-25 25-26 28-29 28-30 30-31

exact/norm bonds :
1-26 2-24 2-25 3-34 9-29 9-31 10-17 10-14 11-35 11-36 12-23 12-24 12-29
13-16 13-17 14-32 16-38 20-35 21-28 28-29 28-30 30-31 39-40 39-41 41-42
43-44 44-45 45-48
exact bonds :
18-33 19-33 22-30 31-38 32-33 33-34 34-35 36-37 37-39 42-43 45-46 46-47
normalized bonds :
4-26 4-27 5-10 6-10 7-13 8-13 15-23 15-27 23-25 25-26

Match level :
1:CLASS 2:Atom 3:CLASS 4:Atom 5:CLASS 6:CLASS 7:CLASS 8:CLASS 9:Atom
10:CLASS 11:CLASS 12:Atom 13:CLASS 14:CLASS 15:Atom 16:CLASS 17:CLASS
18:CLASS 19:CLASS 20:CLASS 21:CLASS 22:CLASS 23:Atom 24:Atom 25:Atom
26:Atom 27:Atom 28:Atom 29:Atom 30:Atom 31:Atom 32:CLASS 33:CLASS 34:CLASS
35:CLASS 36:CLASS 37:CLASS 38:CLASS 39:CLASS 40:CLASS 41:CLASS 42:CLASS
43:CLASS 44:CLASS 45:CLASS 46:CLASS 47:CLASS 48:CLASS

Stereo Bonds:

28-21 (Single Wedge).
30-22 (Single Wedge).
34-3 (Single Wedge).

Stereo Chiral Centers:

28 (Parity=Even)
30 (Parity=Even)
34 (Parity=Odd)

Stereo RSS Sets:

Type=Relative (Default). 3 Nodes= 28 30 34

L1 STRUCTURE UPLOADED

=> s l1
SAMPLE SEARCH INITIATED 10:11:05 FILE 'REGISTRY'
SAMPLE SCREEN SEARCH COMPLETED - 72 TO ITERATE

100.0% PROCESSED 72 ITERATIONS (17 INCOMPLETE) 35 ANSWERS
SEARCH TIME: 00.00.01

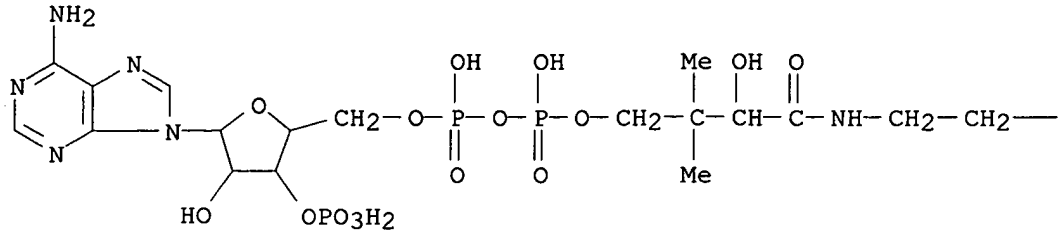
FULL FILE PROJECTIONS: ONLINE **COMPLETE**
BATCH **COMPLETE**
PROJECTED ITERATIONS: 931 TO 1949
PROJECTED ANSWERS: 346 TO 1054

L2 35 SEA SSS SAM L1

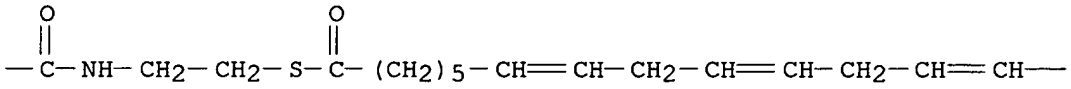
=> d scan

L2 35 ANSWERS REGISTRY COPYRIGHT 2003 ACS on STN
ITERATION INCOMPLETE
IN Coenzyme A, S-7,10,13-hexadecatrienoate, (Z,Z,Z)- (9CI)
MF C37 H60 N7 O17 P3 S

PAGE 1-A



PAGE 1-B



PAGE 1-C

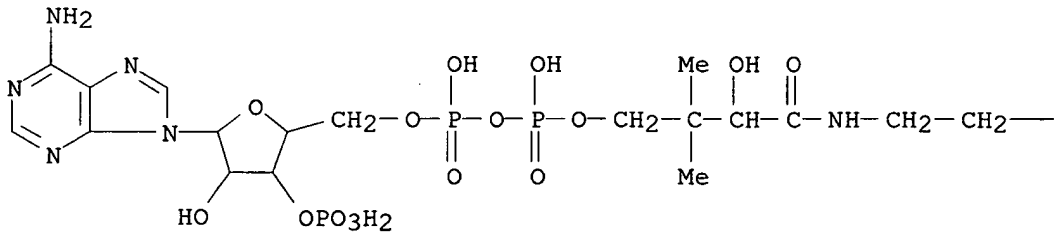
—Et

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

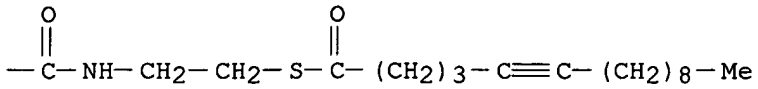
HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):3

L2 35 ANSWERS REGISTRY COPYRIGHT 2003 ACS on STN
ITERATION INCOMPLETE
IN Coenzyme A, S-5-pentadecynoate (9CI)
MF C36 H60 N7 O17 P3 S

PAGE 1-A



PAGE 1-B

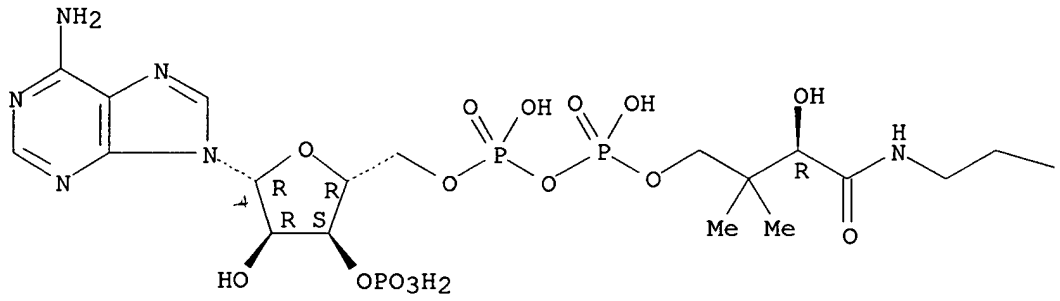


PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

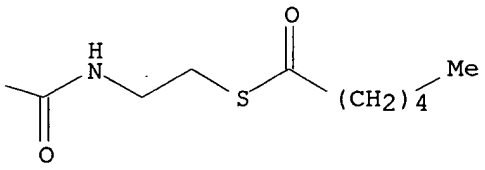
L2 35 ANSWERS REGISTRY COPYRIGHT 2003 ACS on STN
IN Coenzyme A, S-hexanoate (7CI, 8CI, 9CI)
MF C27 H46 N7 O17 P3 S
CI COM

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B

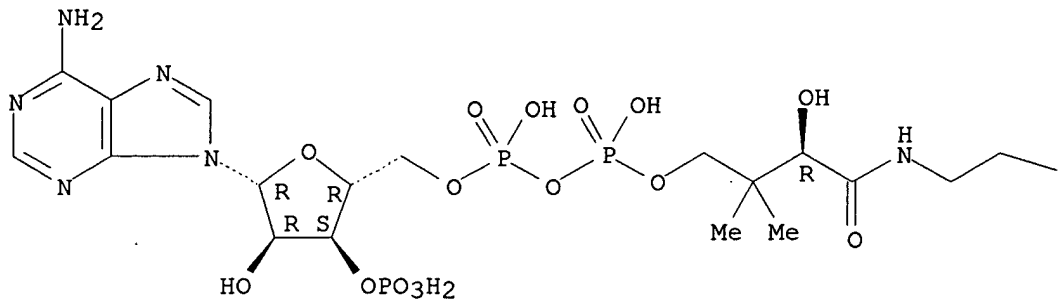


PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

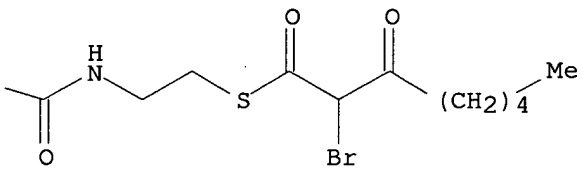
L2 35 ANSWERS REGISTRY COPYRIGHT 2003 ACS on STN
IN Coenzyme A, S-(2-bromo-3-oxooctanoate) (9CI)
MF C29 H47 Br N7 O18 P3 S

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):0

=> s l1 full
FULL SEARCH INITIATED 10:11:49 FILE 'REGISTRY'
FULL SCREEN SEARCH COMPLETED - 1488 TO ITERATE

100.0% PROCESSED 1488 ITERATIONS (313 INCOMPLETE) 643 ANSWERS
SEARCH TIME: 00.00.02

L3 643 SEA SSS FUL L1

=> file caplus

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	148.95	149.16

FILE 'CAPLUS' ENTERED AT 10:12:33 ON 04 OCT 2003
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

FILE COVERS 1907 - 4 Oct 2003 VOL 139 ISS 15
FILE LAST UPDATED: 2 Oct 2003 (20031002/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

```
=> s l3
L4        2435 L3

=> s l4 and (diabetes OR obesity OR PPAR)
         83307 DIABETES
         24320 OBESITY
          3782 PPAR
L5          47 L4 AND (DIABETES OR OBESITY OR PPAR)

=> s l4 and p/dt
       4191595 P/DT
L6          52 L4 AND P/DT

=> s l5 and l6
L7           1 L5 AND L6

=> d

L7   ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2003 ACS on STN
AN   2001:228715  CAPLUS
DN   134:247253
TI   PPAR.alpha. and PPAR.gamma. inhibitors containing
     fatty acid CoA thioesters
IN   Murakami, Koji; Ide, Tomohiro; Mochizuki, Toshiro; Kadowaki, Takashi
PA   Kyorin Pharmaceuticals Co., Ltd., Japan
SO   PCT Int. Appl., 12 pp.
     CODEN: PIXXD2
DT   Patent
LA   Japanese
FAN.CNT 1
     PATENT NO.        KIND  DATE          APPLICATION NO.  DATE
     ---------------   ----  --------      ---------------  --------
PI   WO 2001021181      A1   20010329      WO 1999-JP5217   19990924
         W:  AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU,
             CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL,
             IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD,
             MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK,
             SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY,
             KG, KZ, MD, RU, TJ, TM
         RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE,
             DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF,
             CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
     AU 9957594         A1   20010424      AU 1999-57594    19990924
     EP 1214939         A1   20020619      EP 1999-944810   19990924
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
```

IE, SI, LT, LV, FI, RO, MK, CY, AL
PRAI WO 1999-JP5217 A 19990924
RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s l5 not l7
L8 46 L5 NOT L7

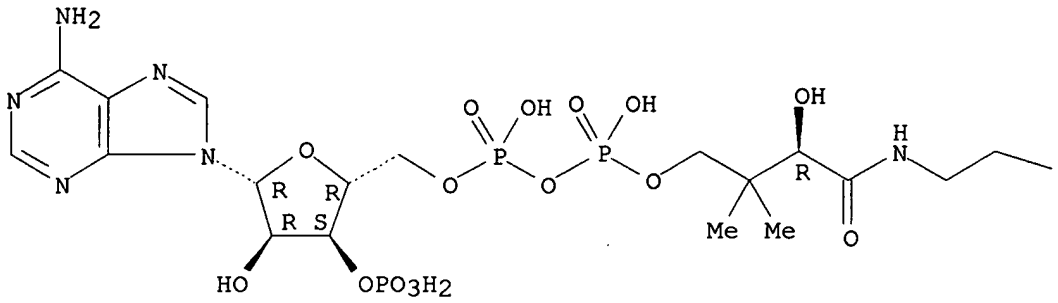
=> sort py
ENTER (L8), L#, OR L# RANGE:.
SORT ENTIRE ANSWER SET? (Y)/N:.
PROCESSING COMPLETED FOR L8
L9 46 SORT L8 PY

=> d 1-20 cbib abs hitstr

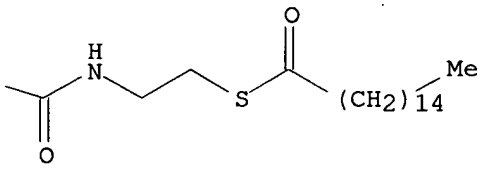
L9 ANSWER 1 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN
1967:497206 Document No. 67:97206 Differential effects of palmitoyl coenzyme A on liver microsomal inorganic pyrophosphate-glucose phosphotransferase and glucose-6-phosphate phosphohydrolase. Nordlie, Robert C.; Hanson, Thomas Lawrence; Johns, Philip T. (Univ. of North Dakota Med. Sch., Grand Forks, ND, USA). Journal of Biological Chemistry, 242(18), 4144-8 (English) 1967. CODEN: JBCHA3. ISSN: 0021-9258.
AB The effects of palmitoyl CoA on both hydrolytic and inorg. pyrophosphate-glucose phosphotransferase activities of glucose-6-phosphate phosphohydrolase (EC 3.1.3.9) of rat liver microsomes were investigated. It was found that the acyl CoA compound may either stimulate or inhibit, in a pH- and concentration- dependent, activity-discriminating manner. Marked activation of the phosphotransferase activity (to a maximum of 350% of control levels in one instance) was observed under a wide variety of conditions, while the glucose-6-phosphate phosphohydrolase activity was at best but modestly elevated (to a maximum of 120% of control levels) and was significantly inhibited by palmitoyl CoA under most conditions tested. A concomitant extensive activation of phosphotransferase and inhibition of phosphohydrolase activity was observed with certain levels of the acyl CoA compound Effective concns. of palmitoyl CoA coincided with established levels of hepatic long chain fatty acyl CoA derivs. CoA, palmitate, and acetyl CoA were without effect on enzymic activities. Maximum phosphotransferase activity was observed at pH 4.3 with untreated microsomal prepns. This maximum was shifted to pH 6 when assays were carried out in the presence of 4.1 + 10-5M palmitoyl CoA, and a significant amount of activity was observed at pH 7.0 under these conditions. It is suggested that changes, in vivo, of functional levels of the hydrolytic and synthetic activities of this enzyme in animals in various altered hormonal states, for example, **diabetes**, possibly may be mediated in part through these activity-discriminating effects of long-chain acyl CoA compds. 31 references.
IT **1763-10-6**
RL: BIOL (Biological study)
(glucose-6-phosphatase hydrolytic and inorg. pyrophosphate-glucose phosphotransferase activities in presence of)
RN 1763-10-6 CAPLUS
CN Coenzyme A, S-hexadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



L9 ANSWER 2 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN
1968:441404 Document No. 69:41404 Inhibition by nucleotides of liver microsomal glucose-6-phosphatase. Nordlie, Robert C.; Hanson, Thomas L.; Johns, Philip T.; Lygre, David G. (Sch. of Med., Univ. of North Dakota, Grand Forks, ND, USA). Proceedings of the National Academy of Sciences of the United States of America, 60(2), 590-7 (English) 1968. CODEN: PNASA6. ISSN: 0027-8424.

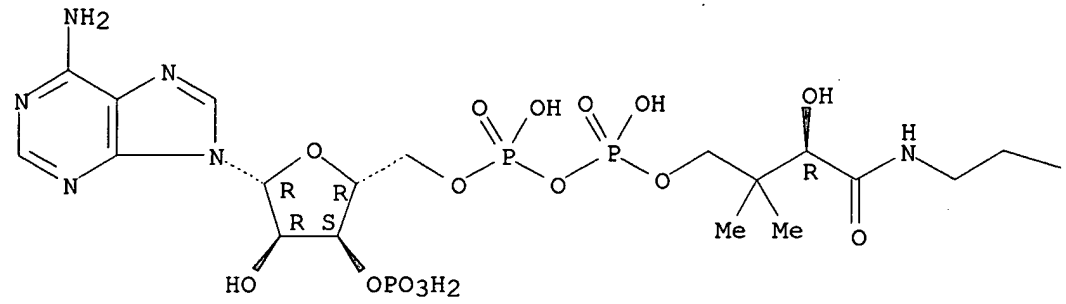
AB ATP, ADP, AMP, CTP, CDP, CMP, ITP, IDP, UTP, UDP, UMP, GTP, GDP, and GMP inhibited competitively the hydrolysis of glucose 6-phosphatase (I) catalyzed by rat liver microsomal glucose-6-phosphatase (II) (EC 3.1.3.9) at pH 5-8, more so at 7.5 and 8.0 than at 6.0-7.0. The inhibition of II activity by nucleotides was usually markedly potentiated by cetyltrimethylammonium bromide (cetrimide) (0.1%, weight/volume in microsomal suspensions), palmityl-CoA (4.1 + 10-5M), and lysolecithin (0.4 mM), suggesting that, although levels of liver II are significantly raised and concns. of ATP and ADP may be moderately reduced in **diabetes**, potentiation by elevated levels of long-chain fatty acyl-CoA esters or other natural detergents of inhibition of III may function to place an ultimate upper limit on the rate of release of liver glucose in diabetic animals in which gluconeogenesis is markedly accelerated. 25 references.

IT **1763-10-6**
RL: BIOL (Biological study)
(glucose 6-phosphatase inhibition by nucleotides in presence of)

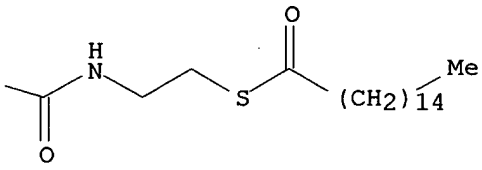
RN 1763-10-6 CAPLUS
CN Coenzyme A, S-hexadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



L9 ANSWER 3 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN
1980:56265 Document No. 92:56265 Training adaptations in skeletal muscle of juvenile diabetics. Costill, D. L.; Cleary, P.; Fink, W. J.; Foster, C.; Ivy, J. L.; Witzmann, F. (Hum. Performance Lab., Ball State Univ., Muncie, IN, 47306, USA). Diabetes, 28(9), 818-22 (English) 1979. CODEN: DIAEAZ. ISSN: 0012-1797.

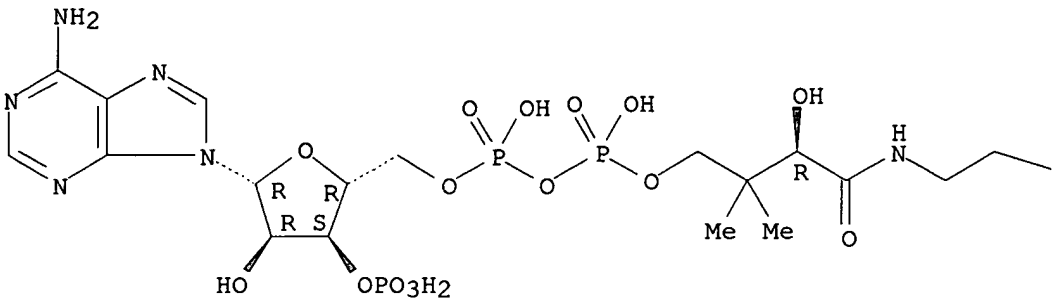
AB Skeletal muscles from male, juvenile-onset diabetics (JD), and nondiabetics (ND) were studied to determine the effects of endurance training on mitochondrial enzyme activities, lipoprotein lipase (LPL) activity, and the oxidation of lipids (palmityl CoA-14C) in vitro. Ten wk of endurance running (30 min/day, 5 days/wk) resulted in 11.0 and 12.9% gains in aerobic capacity for the JD and ND groups, resp. Both groups showed significant increases in muscle LPL, carnitine palmityl transferase, succinate dehydrogenase, and hexokinase activities with training. Though the pretraining capacities for palmityl CoA-14C oxidation were similar for both ND and JD groups, the diabetics showed a 41% greater improvement in the measurement of muscle lipid oxidation after training than did the ND group. Thus, skeletal muscle of juvenile diabetics who are in moderate insulin balance shows adaptations to endurance training that are similar to those of nondiabetic men.

IT **1763-10-6**
RL: RCT (Reactant); RACT (Reactant or reagent)
(oxidation of, by muscle mitochondria of juvenile diabetics, endurance training in relation to)

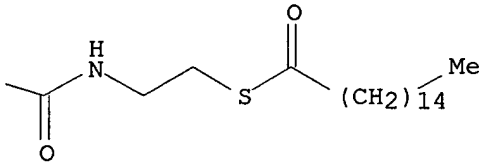
RN 1763-10-6 CAPLUS
CN Coenzyme A, S-hexadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



L9 ANSWER 4 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN

1979:436368 Document No. 91:36368 Regulation of hepatic fatty acid oxidation and esterification. Mannaerts, Guy P.; Debeer, Luc J. (Sch. Med., Kathol. Univ. Leuven, Leuven, B-3000, Belg.). Developments in Endocrinology (Amsterdam), Volume Date 1978, 4(Lipoprotein Metab. Endocr. Regul.), 271-8 (English) 1979. CODEN: DENDD4. ISSN: 0165-1900.

AB The rates of mitochondrial and peroxisomal palmitoyl-CoA oxidation (in whole liver homogenates) were compared at low free substrate concns., obtained by the addition of various amts. of albumin to the incubation mixts. At all substrate/albumin ratios tested, peroxisomal oxidation was lower than mitochondrial oxidation except when no albumin was present. Mitochondrial oxidation was severely impaired in the absence of albumin. The peroxisomal oxidation was CoA and NAD dependent and mitochondrial oxidation was carnitine dependent. Mitochondrial oxidation was strongly inhibited by (+)-octanoylcarnitine and malonyl-CoA, whereas peroxisomal oxidation was unaffected. The rate of peroxisomal fatty acid oxidation was unaffected by starvation and **diabetes**, although both conditions are characterized by an increased capacity of the liver to oxidize long-chain fatty acids. The rates of oleate and palmitate oxidation in isolated, intact hepatocytes were inhibited by 87% and 75%, resp., with 5 mM (+)-octanoylcarnitine and by 80% with 2 mM KCN. Esterification was not affected by KCN. Apparently, in the normal liver the bulk of fatty acid oxidation is mitochondrial. Peroxisomal oxidation may become important when long-chain fatty acid-CoA would accumulate in the cell because of its lower substrate affinity.

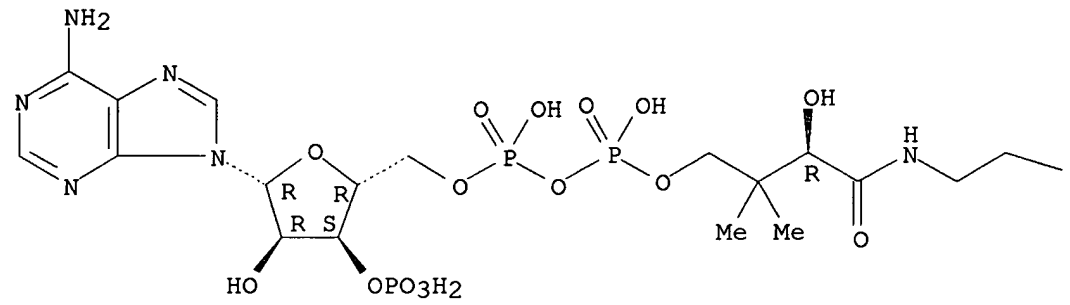
IT **1763-10-6**
RL: RCT (Reactant); RACT (Reactant or reagent)
(oxidation of, by liver mitochondria and peroxisome)

RN 1763-10-6 CAPLUS

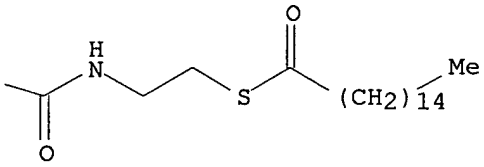
CN Coenzyme A, S-hexadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A

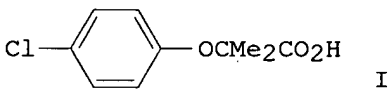


PAGE 1-B



L9 ANSWER 5 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN
1983:46752 Document No. 98:46752 Increased activity of stearoyl-CoA desaturation in liver from rat fed clofibric acid. Kawashima, Yoichi; Kozuka, Hiroshi (Fac. Pharm. Sci., Toyama Med. Pharm. Univ., Toyama, 930-01, Japan). Biochimica et Biophysica Acta, 713(3), 622-8 (English) 1982. CODEN: BBACAQ. ISSN: 0006-3002.

GI



I

AB Male rats were fed a diet containing 0.5% clofibric acid (I) [882-09-7], a hypolipidemic drug. stearoyl-CoA [**362-66-3**] Desatn. in hepatic microsomes were increased approx. 4 times following the administration of clofibric acid for 7 days. An increase in the desatn. of stearic acid [57-11-4] was also observed in the liver of clofibric acid-fed rats in vivo. The increase in the microsomal stearoyl-CoA desatn. by clofibric acid-feeding was due to the increase in the activity of terminal desaturase as measured by the rate constant for cytochrome b5 reoxidn., but not due to the changes in cytochrome b5 content and NADH-cytochrome b5 reductase activity. Increases in the stearoyl-CoA desatn. by clofibric acid-feeding were also observed in rats with hormonal disorders (**diabetes**, hypo- and hyperthyroid function). Percentages of

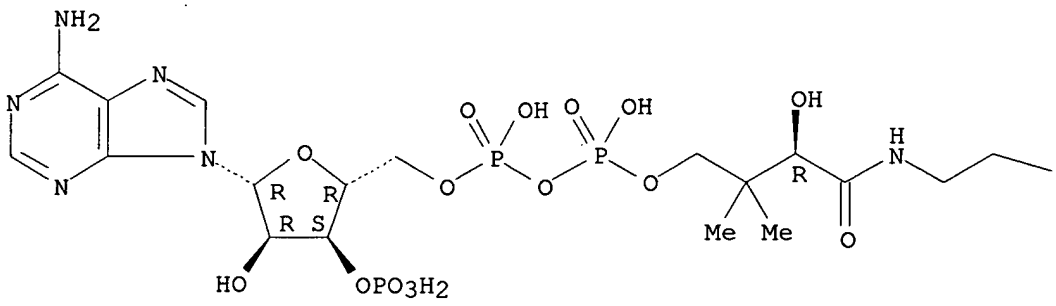
octadecenoic acid in total fatty acid of hepatic lipid were increased with the increase in the activity of stearoyl-CoA desatn.

IT **362-66-3**
RL: BIOL (Biological study)
(desatn. of, by liver microsomes, clofibric acid effect on)

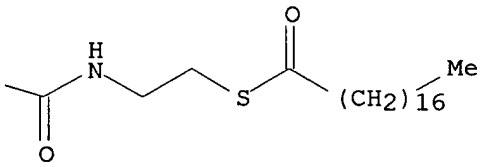
RN 362-66-3 CAPLUS
CN Coenzyme A, S-octadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



L9 ANSWER 6 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN
1982:402737 Document No. 97:2737 Regulation of the biosynthesis of CoA at the level of pantothenate kinase. Halvorsen, Ola; Skrede, Sverre (Inst. Clin. Biochem., Rikshosp., Oslo, Norway). European Journal of Biochemistry, 124(1), 211-15 (English) 1982. CODEN: EJBCAI. ISSN: 0014-2956.

AB Pantothenate kinase (I) which is present in cytosol, was studied in prepns. from livers of rats fed normal or clofibrate-enriched diets. The effects of CoA, dephospho-CoA, and different acyl-CoA derivs. on I were examined in vitro. With partially purified I or crude particle-free supernatant from the liver of normal or clofibrate-treated rats, the Km for pantothenic acid was 0.016 mM at the pH optimum of 6.1. Acetyl-CoA, propionyl-CoA, malonyl-CoA, and other short-chain acyl-CoA derivs. were strong inhibitors of I with Ki values in the range 0.001-0.003 mM. The mechanism of inhibition appeared to be of an uncompetitive type. Free CoA, dephospho-CoA, and long-chain acyl-CoA (with Ki 0.003-0.08 mM) were less efficient inhibitors than acetyl-CoA. With I from clofibrate-treated animals, all inhibitors were less potent. This was most pronounced when I was assayed in a crude supernatant fraction, possibly because the inhibitors were degraded and/or protein bound. Such a reduction of normal

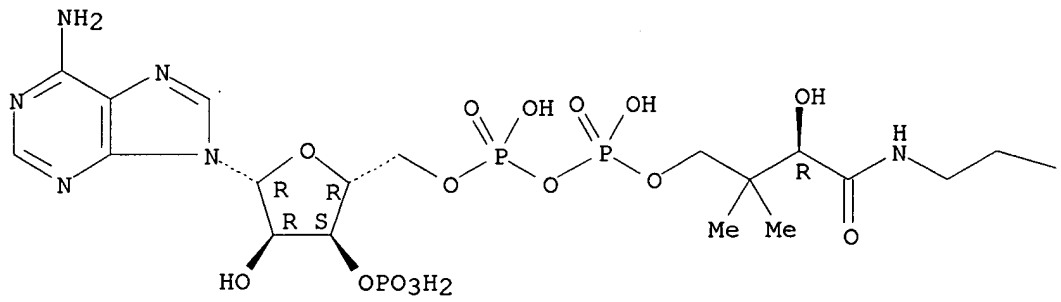
inhibition may contribute to the increased biosynthesis of CoA previously observed during clofibrate treatment. Fasting or **diabetes** leads to an increase of long-chain acyl-CoA and total CoA in the liver. The increase of CoA has previously been explained by increased acylation of CoA, and thereby reduced feedback inhibition by free CoA at the I level. Another explanation is proposed here. In these metabolic states, the cytosolic pool of acetyl-CoA is decreased. As I is present only in the cytosol, its activity will be released and the biosynthesis of CoA will increase. Acetyl-CoA is probably a more important physiol. regulator of I activity than is free CoA.

IT **1264-52-4 1716-06-9 1763-10-6**
17046-56-9
RL: BIOL (Biological study)
(pantothenate kinase inhibition by)

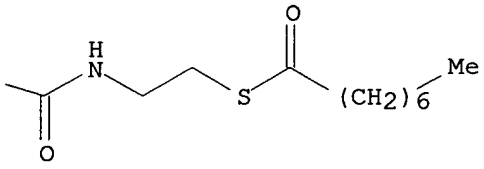
RN 1264-52-4 CAPLUS
CN Coenzyme A, S-octanoate (6CI, 7CI, 8CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



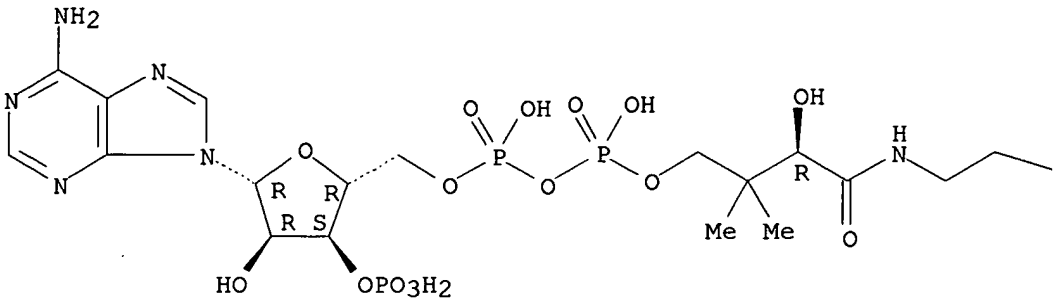
PAGE 1-B



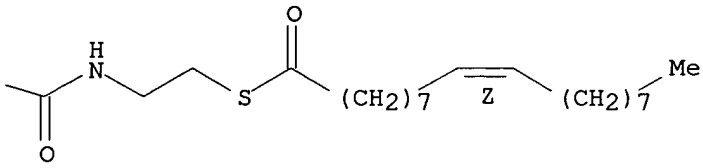
RN 1716-06-9 CAPLUS
CN Coenzyme A, S-(9Z)-9-octadecenoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

PAGE 1-A



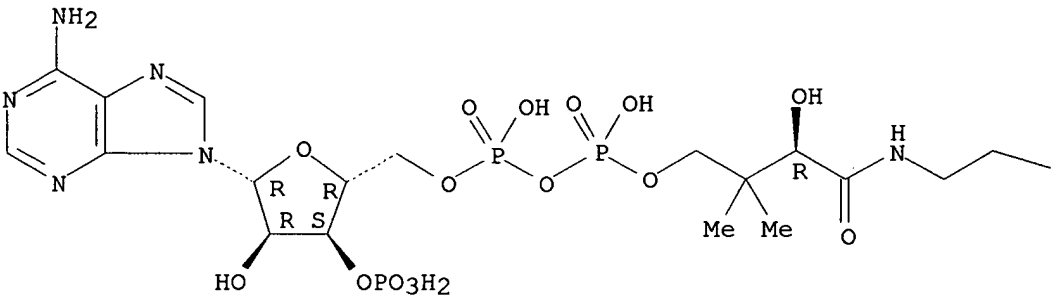
PAGE 1-B



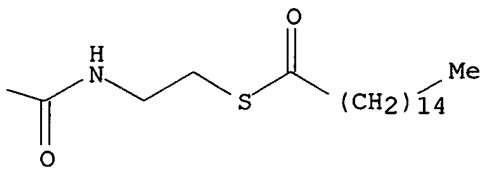
RN 1763-10-6 CAPLUS
CN Coenzyme A, S-hexadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



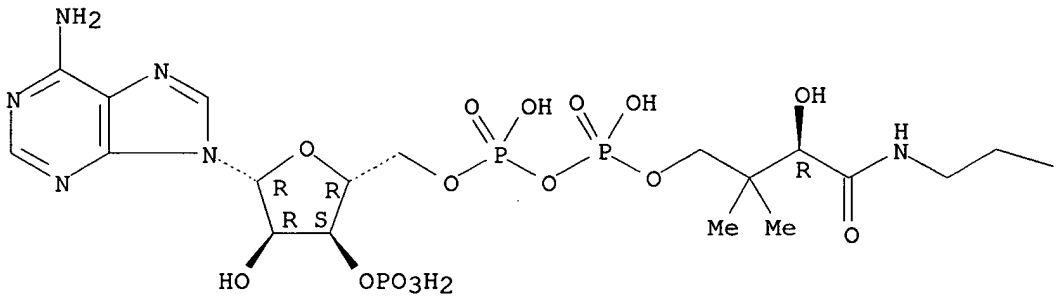
PAGE 1-B



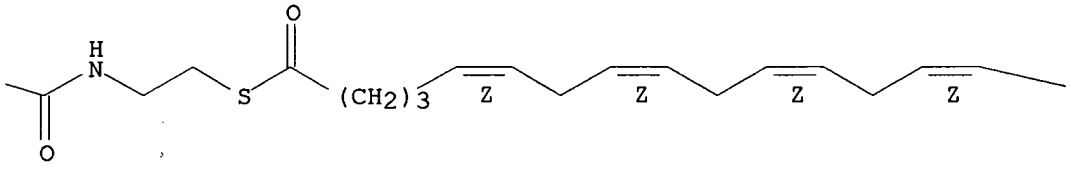
RN 17046-56-9 CAPLUS
CN Coenzyme A, S-(5Z,8Z,11Z,14Z)-5,8,11,14-eicosatetraenoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

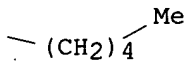
PAGE 1-A



PAGE 1-B



PAGE 1-C



L9 ANSWER 7 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN
1983:556397 Document No. 99:156397 Hepatic mitochondrial function in lean and obese Zucker rats. Brady, Linda J.; Hoppel, Charles L. (Veterans Adm. Med. Cent., Cleveland, OH, 44106, USA). American Journal of Physiology, 245(3), E239-E245 (English) 1983. CODEN: AJPHAP. ISSN: 0002-9513.

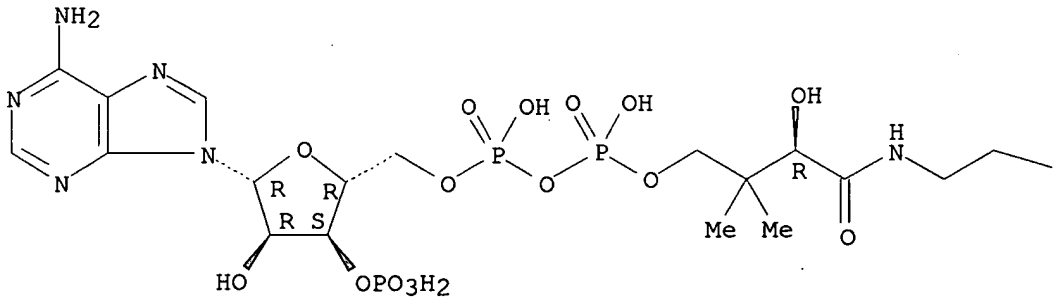
AB Hepatic mitochondrial function was studied in lean and obese Zucker rats in the fed state and at 3 and 6 days of starvation. No differences in state 3 mitochondrial oxidative rates were found due to **obesity** or starvation. Palmitoylcarnitine utilization rates in mitochondria were unaffected by **obesity** or starvation; however, when expressed per g liver weight, they were lower in the obese rats due to the decreased amount of mitochondrial protein per g liver. For palmitoylcarnitine oxidation and acetoacetate and citrate production, the patterns were the same: per mg mitochondrial protein, both lean and obese rates were equivalent; per total liver, the obese rates were higher; per g liver, the obese rates were lower. Mitochondrial carnitine palmitoyltransferase specific activity was high in fed obese than in lean rats and remained higher during starvation. Mitochondrial capacity to oxidize fatty acids and to produce keto acids is not affected by genetic **obesity** or starvation. The differences in fatty acid oxidation and keto acid production that have been observed in hepatocytes and perfused liver might be explained by decreased mitochondrial protein per unit weight of liver or hepatocytes in obese rats.

IT **1264-52-4**
RL: RCT (Reactant); RACT (Reactant or reagent)
(oxidation of, by liver mitochondria in genetic **obesity** and starvation, mitochondrial proteins in relation to)

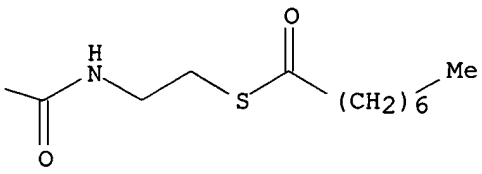
RN 1264-52-4 CAPLUS
CN Coenzyme A, S-octanoate (6CI, 7CI, 8CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



L9 ANSWER 8 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN
1983:177214 Document No. 98:177214 Fatty acid utilization and purine nucleotide binding in brown adipose tissue of genetically obese (ob/ob) mice. Bas, Sylvette; Imesch, Elisabeth; Ricquier, Daniel; Assimacopoulos-Jeannet, Francoise; Seydoux, Josiane; Giacobino, Jean Paul (Dep. Biochim. Med., Cent. Med. Univ., Geneva, 1211, Switz.). Life Sciences, 32(18), 2123-30 (English) 1983. CODEN: LIFSAK. ISSN: 0024-3205.

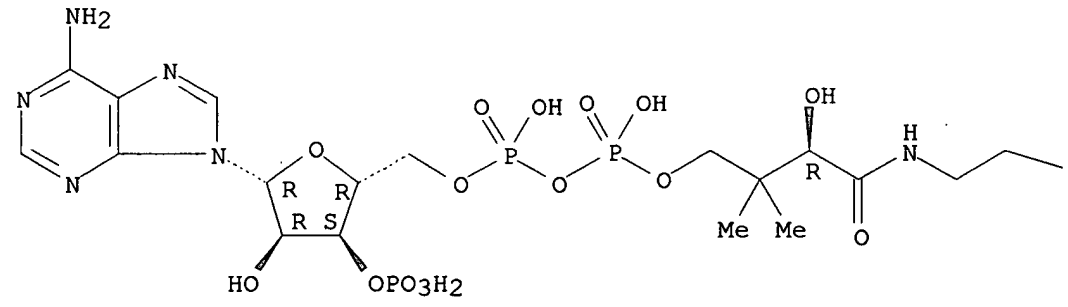
AB In ob/ob mice at 23°, the specific activities of the palmitoyl CoA-synthetase and of palmitoyl-CoA β-oxidation as well as the number of GDP binding sites were lower than in the lean mice by 26, 43, and 37%, resp. The percentage of total mitochondrial protein comprising 32,000-mol.-weight protein, which participates in the thermogenic H+ conductance pathway and binds GDP, was the same in both groups. In the ob/ob mice at 23°, the lower homogenate β-oxidation specific activity was due to the fact that the peroxisomal and mitochondrial specific activities were 44 and 37% lower, resp. Cold acclimation at 4° caused an increase of the palmitoyl-CoA synthetase specific activity, of the palmitoyl-CoA synthetase and peroxisomal β-oxidation total activities, and of the number of GDP binding sites, in both lean and ob/ob mice. Cold acclimation increased the percentage of 32,000 polypeptide in the ob/ob mice only.

IT **1763-10-6**
RL: BIOL (Biological study)
(β-oxidation by mitochondria and peroxisomes of brown adipose tissue, of genetically obese mice in cold adaptation)

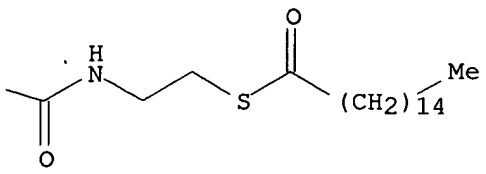
RN 1763-10-6 CAPLUS
CN Coenzyme A, S-hexadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



L9 ANSWER 9 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN
1985:452107 Document No. 103:52107 Factors that influence myocardial levels of long-chain acyl CoA and acyl carnitine. Neely, J. R.; McDonough, K. H. (Dep. Physiol., Milton S. Hershey Med. Cent., Hershey, PA, 17033, USA). Myocard. Ischemia Lipid Metab., [Proc. Int. Soc. Heart Res. Myocard. Ischemia Lipid Metab.], Meeting Date 1983, 159-69. Editor(s): Ferrari, Roberto. Plenum: New York, N. Y. (English) 1984. CODEN: 53QPA7.

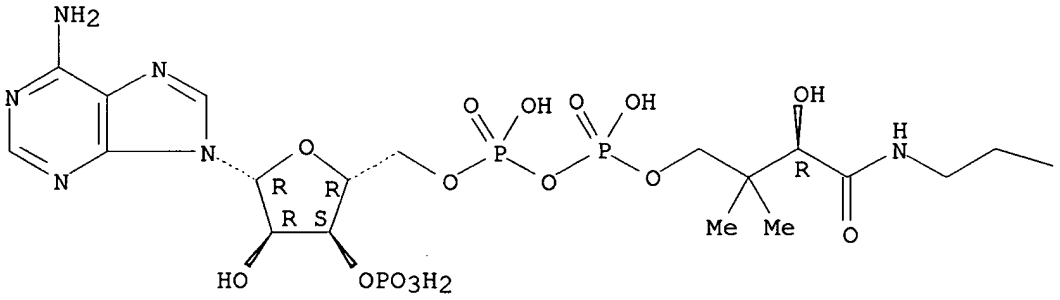
AB Fatty acyl CoA and fatty acyl carnitine levels in isolated perfused rat hearts were increased by fasting, **diabetes** mellitus, and especially ischemia. The ischemia-associated accumulation of these esters did not alter the ability of the heart to resynthesize ATP or creatine phosphate or to regain mech. function with reperfusion. Palmitoyl-CoA inhibited cardiac triglyceride lipase, whereas palmitoyl carnitine and palmitate were much weaker enzyme inhibitors. The role of lipase inhibition by palmitoyl-CoA in the triglyceride accumulation in ischemic and diabetic hearts is briefly discussed.

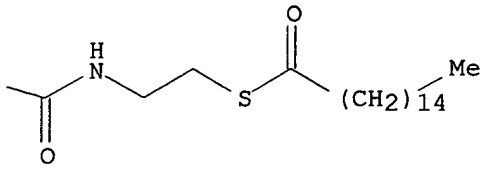
IT **1763-10-6**
RL: BIOL (Biological study)
(triglyceride lipase inhibition by, heart ischemia in relation to)

RN 1763-10-6 CAPLUS
CN Coenzyme A, S-hexadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A





L9 ANSWER 10 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN
1984:608600 Document No. 101:208600 Effects of streptozotocin-induced **diabetes** on phosphoglyceride metabolism of the rat liver. Dang, An Quoc; Faas, Fred H.; Carter, William J. (Veterans Adm. Med. Cent., Little Rock, AR, 72206, USA). Lipids, 19(10), 738-48 (English) 1984. CODEN: LPDSAP. ISSN: 0024-4201.

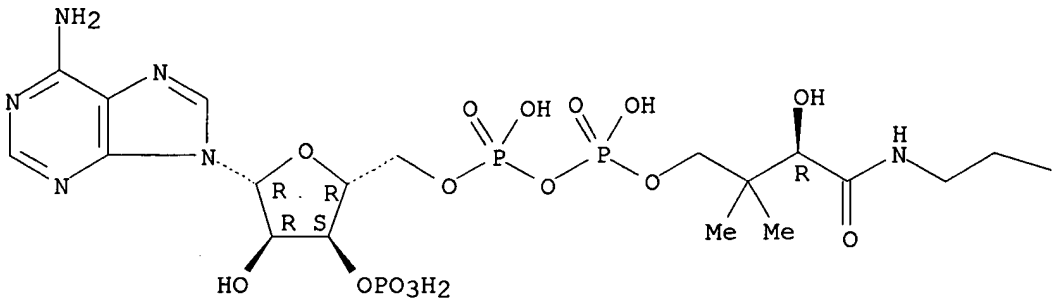
AB The effect of streptozotocin (SZ)-induced **diabetes** on fatty acyltransferase and phospholipase enzyme activities involved in the synthesis and degradation of rat liver phosphoglycerides was studied. Neither mitochondrial nor microsomal acyl-CoA:glycerol 3-phosphate acyltransferase (GPAT) activity was altered, although insulin treatment stimulated mitochondrial GPAT activity. However, microsomal acyl-CoA:1-acylglycerol 3-phosphate acyltransferase (1-acyl-GPAT) activity increased (24-33 per cent, $p < 0.01$) in the diabetic animals using 3 different acyl-CoA donors: palmitoyl-CoA, oleoyl-CoA and linoleoyl-CoA. SZ-induced **diabetes** also increased acyl-CoA:1-acylglycerol 3-phosphorylcholine acyltransferase (GPCAT) activity (38-45 per cent, $p < 0.01$) with 3 different acyl-CoA donors: oleoyl-CoA, linoleoyl-CoA, and arachidonoyl-CoA. 1-Acyl-GPAT and GPCAT activity returned to normal with insulin treatment. In contrast to the increased activity of the microsomal fatty acyltransferases 1-acyl-GPAT and GPCAT, SZ-induced **diabetes** decreased mitochondrial phospholipase A2 activity and lysophospholipase activity (49-70 per cent, $p < 0.01$). Insulin treatment of the diabetic rats corrected the decreased lysophospholipase and stimulated phospholipase A2 activity 35 per cent higher than controls. Since microsomal 1-acyl-GPAT and GPCAT are known to have higher activity toward unsatd. fatty acyl-CoA donors, the increased GPCAT activity coupled with the decreased lysophospholipase activity and the increased 1-acyl-GPAT activity in **diabetes** would tend to increase the formation of newly synthesized phospholipids containing unsatd. fatty acids. This mechanism plus the decreased fatty acid desaturase may be the factors which alter the fatty acid composition of phosphoglycerides in diabetic rat liver microsomes.

IT **1716-06-9 1763-10-6 6709-57-5**
17046-56-9
RL: PROC (Process)
(incorporation of, into phosphoglycerides by liver in **diabetes** mellitus)

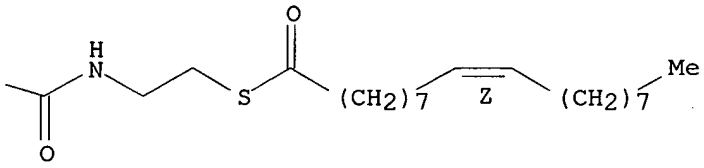
RN 1716-06-9 CAPLUS
CN Coenzyme A, S-(9Z)-9-octadecenoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

PAGE 1-A



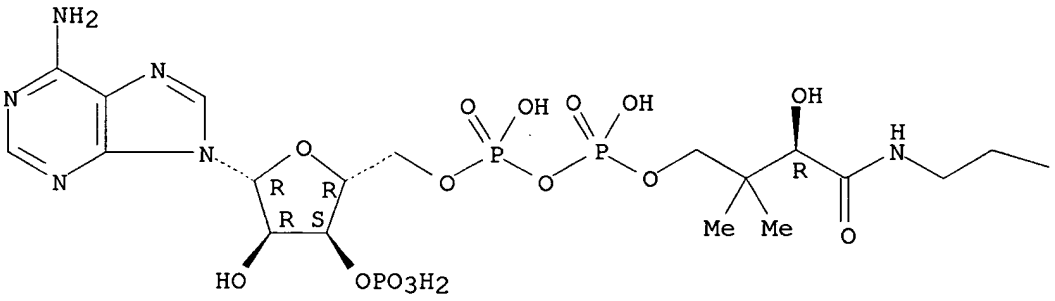
PAGE 1-B



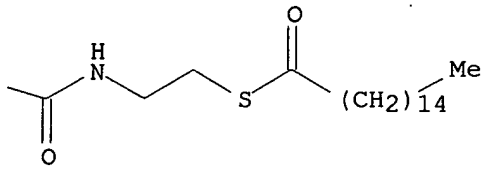
RN 1763-10-6 CAPLUS
CN Coenzyme A, S-hexadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



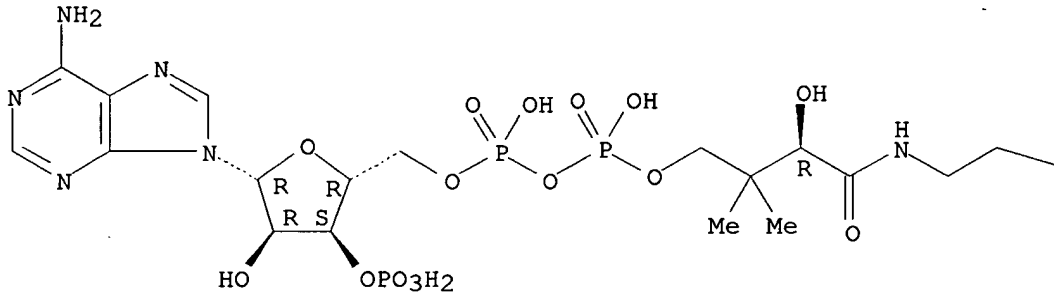
PAGE 1-B



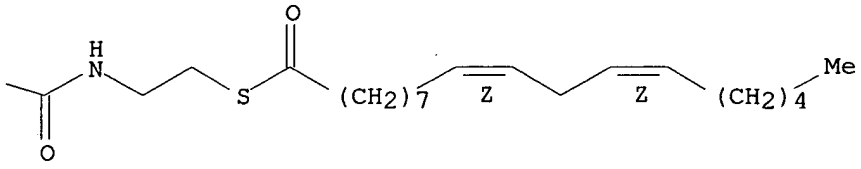
RN 6709-57-5 CAPLUS
CN Coenzyme A, S-(9Z,12Z)-9,12-octadecadienoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

PAGE 1-A



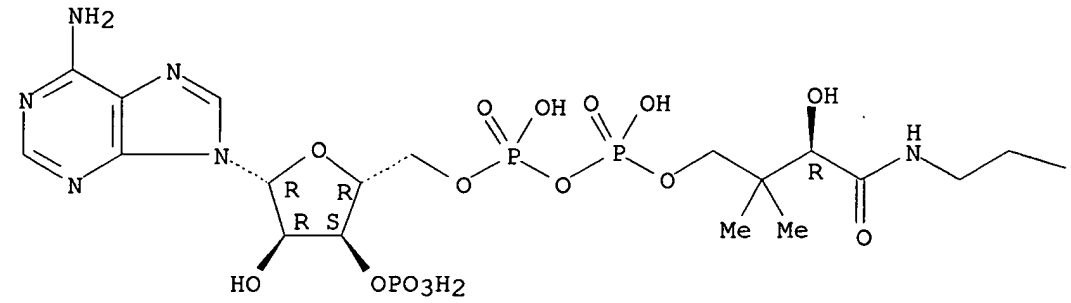
PAGE 1-B



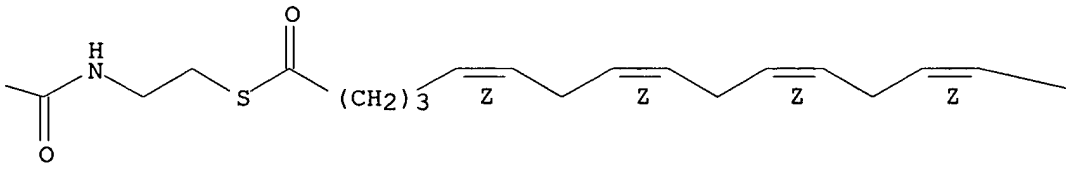
RN 17046-56-9 CAPLUS
CN Coenzyme A, S-(5Z,8Z,11Z,14Z)-5,8,11,14-eicosatetraenoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

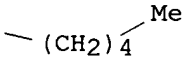
PAGE 1-A



PAGE 1-B



PAGE 1-C



L9 ANSWER 11 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN

1986:204978 Document No. 104:204978 The effect of palmitoyl-CoA on GDP binding to brown adipose tissue mitochondria from lean and obese Zucker rats. Haggarty, Paul; Percival, Susannah (Director's Unit, Towett Res. Inst., Aberdeen, AB2 9SB, UK). Biochemical Society Transactions, 14(2), 290-1 (English) 1986. CODEN: BCSTB5. ISSN: 0300-5127.

AB The maximum GDP binding capacity and GDP binding affinity of brown adipose tissue mitochondria (BAT-m) from lean 35-day-old Zucker rats was greater than that of age-matched obese rats. Addition of palmitoyl-CoA (1 mmol/mg protein) reduced the maximum GDP binding capacity and binding affinity in both phenotypes. BAT-m from obese animals contained significantly more fatty acyl-CoA compared to lean rats. That the phenotypic difference in GDP binding may be partly explained by the higher levels of fatty acyl-CoA in obese rat BAT-m is suggested.

IT **1763-10-6**
RL: BIOL (Biological study)

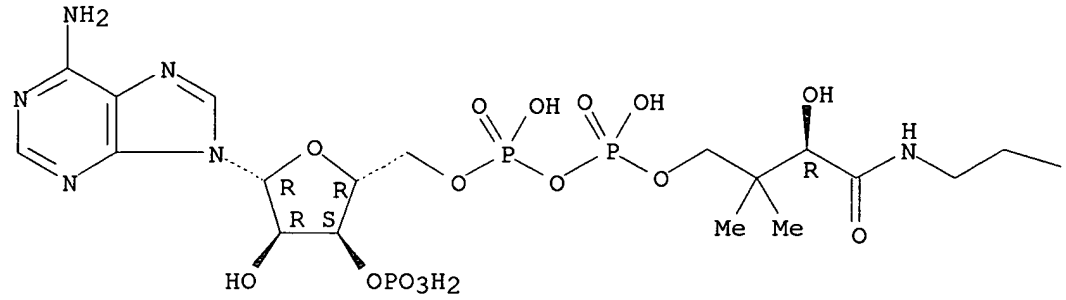
(GDP binding response to, of brown adipose tissue mitochondria of obese Zucker rat)

RN 1763-10-6 CAPLUS

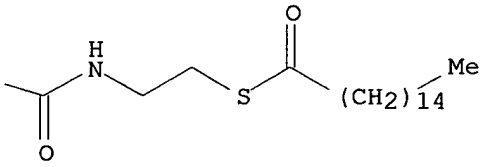
CN Coenzyme A, S-hexadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



L9 ANSWER 12 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN

1986:86370 Document No. 104:86370 Fatty acid specificity of acyl-CoA synthetase in rat glomeruli. Morisaki, Nobuhiro; Kanzaki, Tetsuto; Saito, Yasushi; Yoshida, Sho (Sch. Med., Chiba Univ., Chiba, 280, Japan). Biochimica et Biophysica Acta, 875(2), 311-15 (English) 1986. CODEN: BBACAQ. ISSN: 0006-3002.

AB The fatty acid specificity of acyl-CoA synthetase in rat glomeruli for physiol. and pathol. important long-chain fatty acids was studied. The apparent Km for substrate fatty acids increased in the order, linolenic < linoleic < eicosapentaenoic < arachidonic < oleic < palmitic acid. The maximum velocities with these fatty acids decreased in the order, oleic > linoleic > palmitic (≈) linolenic > arachidonic > eicosapentaenoic acid. The syntheses of radioactive arachidonyl-CoA and palmitoyl-CoA from radioactive arachidonic and palmitic acid, resp., were both inhibited by all fatty acids mentioned above including the substrate fatty acids, their inhibitory effects being inversely correlated with their apparent Km values. Apparently, the enzyme in glomeruli has a unique specificity for fatty acids and there is no arachidonic acid-specific acyl-CoA synthetase in glomeruli. The possible contribution of the glomerular enzyme with this specificity to the abnormal fatty acid levels in diabetic animals is discussed.

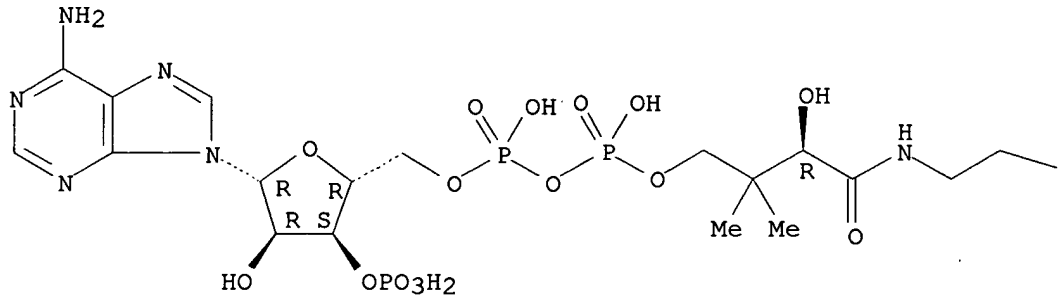
IT **1716-06-9 1763-10-6 6709-57-5 17046-56-9**

RL: FORM (Formation, nonpreparative)
(formation of, by kidney glomerulus, fatty acid inhibition of, **diabetes** mellitus in relation to)

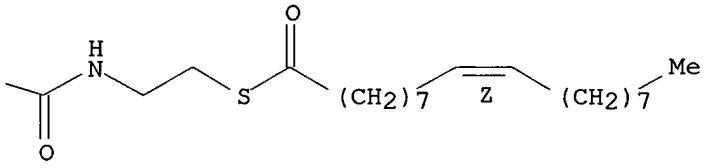
RN 1716-06-9 CAPLUS
CN Coenzyme A, S-(9Z)-9-octadecenoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

PAGE 1-A



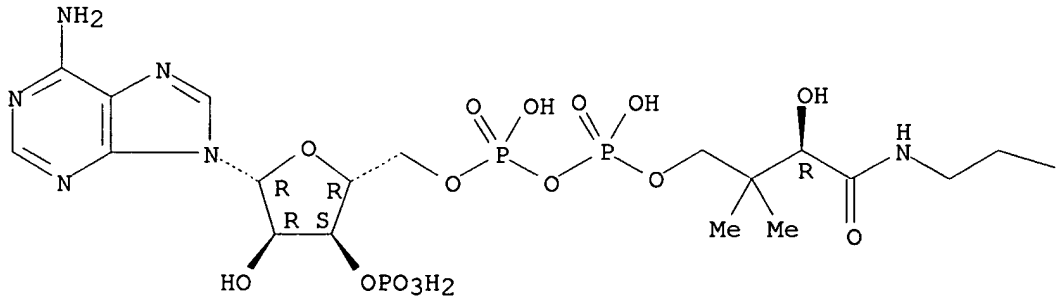
PAGE 1-B



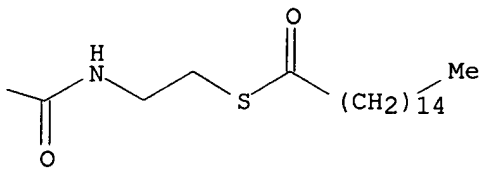
RN 1763-10-6 CAPLUS
CN Coenzyme A, S-hexadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



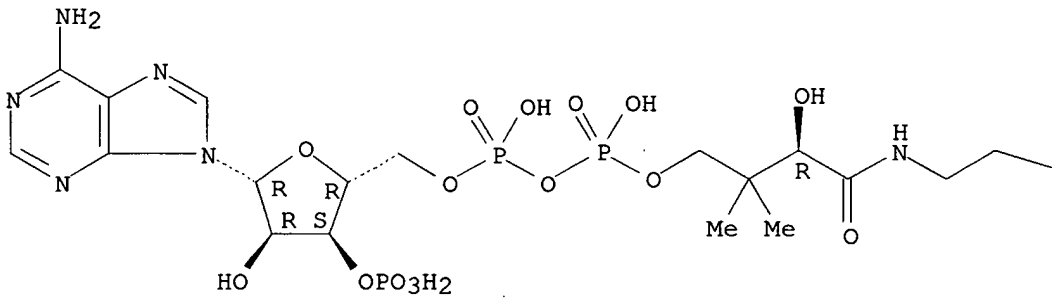
PAGE 1-B



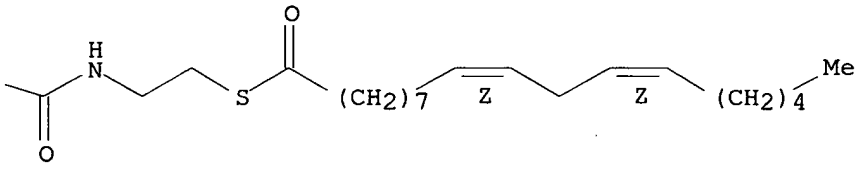
RN 6709-57-5 CAPLUS
CN Coenzyme A, S-(9Z,12Z)-9,12-octadecadienoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

PAGE 1-A



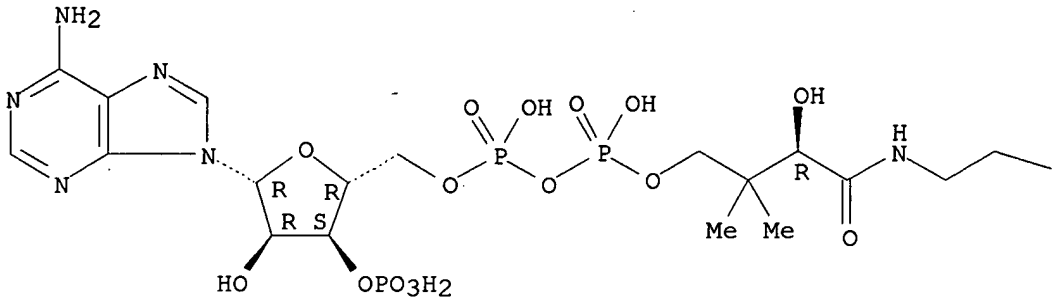
PAGE 1-B



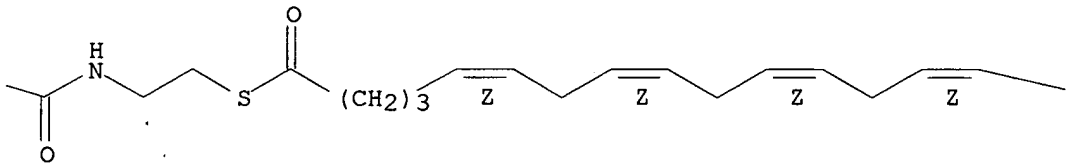
RN 17046-56-9 CAPLUS
CN Coenzyme A, S-(5Z,8Z,11Z,14Z)-5,8,11,14-eicosatetraenoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

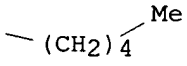
PAGE 1-A



PAGE 1-B



PAGE 1-C



L9 ANSWER 13 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN
1987:174008 Document No. 106:174008 Effect of starvation and **diabetes** on the sensitivity of carnitine palmitoyltransferase I to inhibition by 4-hydroxyphenylglyoxylate. Stephens, Thomas W.; Harris, Robert A. (Sch. Med., Indiana Univ., Indianapolis, IN, 46223, USA). Biochemical Journal, 243(2), 405-12 (English) 1987. CODEN: BIJOAK. ISSN: 0306-3275.

AB The sensitivity of carnitine palmitoyltransferase I to inhibition by 4-hydroxyphenylglyoxylate was decreased markedly in liver mitochondria isolated from either 48 h-starved or streptozotocin-diabetic rats. These treatments of the rat also decreased the sensitivity of fatty acid oxidation by isolated hepatocytes to inhibition by this compound Incubation of hepatocytes prepared from fed rats with N6O2'-dibutyryl cAMP also decreased the sensitivity, whereas incubation of hepatocytes prepared from starved rats with lactate plus pyruvate had the opposite effect on 4-hydroxyphenylglyoxylate inhibition of fatty acid oxidation The sensitivity

of carnitine palmitoyltransferase I of mitochondria to 4-hydroxyphenylglyoxylate increased in a time-dependent manner, as previously reported for malonyl-CoA. Likewise, oleoyl-CoA activated carnitine palmitoyltransferase I in a time-dependent manner and prevented the sensitization by 4-hydroxyphenylglyoxylate. Increased exogenous carnitine caused a moderate increase in fatty acid oxidation by hepatocytes under some conditions and a decreased 4-hydroxyphenylglyoxylate inhibition of fatty acid oxidation at low oleate concentration, without decreasing the difference in 4-hydroxyphenylglyoxylate inhibition between fed- and starved-rat hepatocytes. Time-dependent changes in the conformation of carnitine palmitoyltransferase I or the membrane environment may be involved in differences among nutritional states in 4-hydroxyphenylglyoxylate-sensitivity of carnitine palmitoyltransferase I.

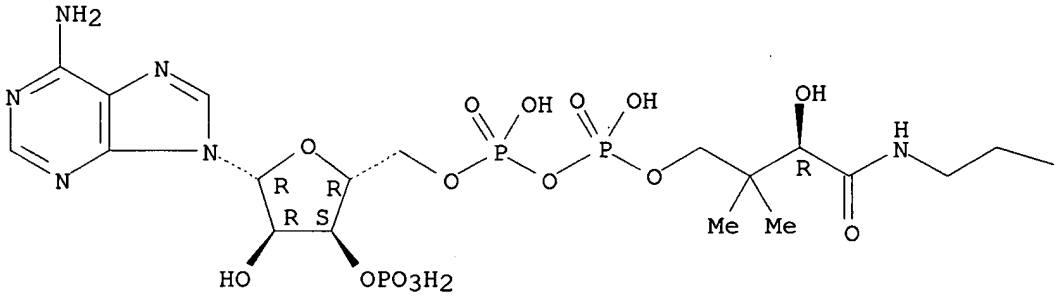
IT **1716-06-9**, Oleoyl-CoA
RL: BIOL (Biological study)
(carnitine palmitoyltransferase I activation response to, in **diabetes** mellitus and starvation, hyroxyphenylglyoxylate inhibition in relation to)

RN 1716-06-9 CAPLUS

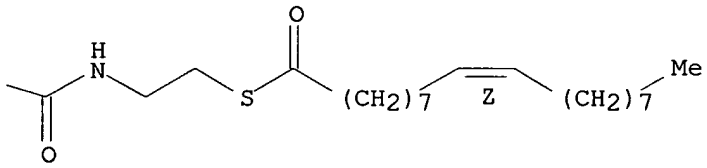
CN Coenzyme A, S-(9Z)-9-octadecenoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

PAGE 1-A



PAGE 1-B



L9 ANSWER 14 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN

1988:588229 Document No. 109:188229 Carnitine palmitoyltransferase: effects of **diabetes**, fasting, and pH on the reaction that generates acyl CoA. McCormick, Kenneth; Mick, Gail J.; Mattson, Viviann; Saile, Debra; Starr, Donald (Health Sci. Cent., SUNY, Syracuse, NY, 13210, USA). Metabolism, Clinical and Experimental, 37(11), 1073-7 (English) 1988.

CODEN: METAAJ. ISSN: 0026-0495.

AB Although carnitine palmitoyltransferase (CPT) has received considerable attention, particularly its regulation by malonyl CoA, most studies have monitored the forward reaction, i.e., the formation of acylcarnitine. The authors studied the reverse reaction, in which palmitoyl CoA is generated, in osmotically-disrupted rat hepatic mitochondria. Specifically, the effects of pH, fasting, and untreated recent-onset **diabetes** were investigated. As with the forward (f) reaction, the CPT reverse (r) velocity vs. pH curve was somewhat parabolic with a pH maximum at .apprx.7.2 (except the CPT that was from the diabetic rats). However, as the pH rose, the CPT reverse and forward curves diverged due to a precipitous decline in the forward reaction. This discordance in rates in the alkaline range was apparent in all 3 groups of CPT but was most prominent in the diabetic preparation, e.g., as the pH increased from 7.3 to 8.8, the resp. declines in the f and r velocities were 74% and 2%. In addition, under these assay conditions the CPTr from diabetic rats not only had a higher velocity than that from the fed or fasted animals, but also the Vmax was 2-fold greater, even though there was no difference in the Km for palmitoylcarnitine. In summary, **diabetes** affects the kinetics of the reverse reaction, and regardless of the animal's premortem condition, but more so in the **diabetes**, this reaction is less attenuated than the forward one as the pH rises.

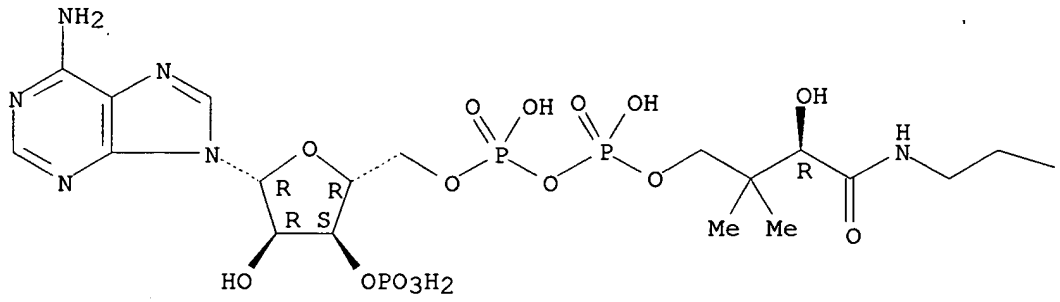
IT **1763-10-6**, Palmitoyl CoA
RL: BIOL (Biological study)
(**diabetes** and fasting and pH effect on formation of, carnitine palmitoyltransferase in relation to)

RN 1763-10-6 CAPLUS

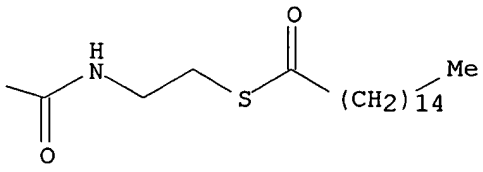
CN Coenzyme A, S-hexadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



L9 ANSWER 15 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN
1988:219848 Document No. 108:219848 Changes in brown-adipose-tissue mitochondrial processes in streptozotocin-**diabetes**. Jamal, Zahirali; Saggerson, E. David (Dep. Biochem., Univ. Coll. London, London, WC1E 6BT, UK). Biochemical Journal, 252(1), 293-6 (English) 1988. CODEN: BIJOAK. ISSN: 0306-3275.

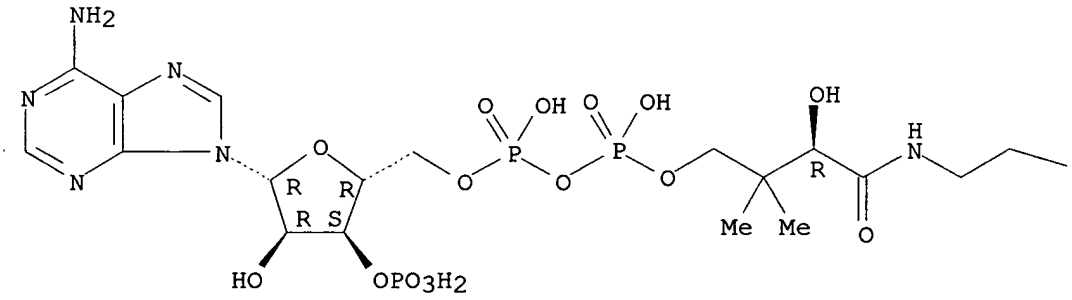
AB Diabetic rats were used as a source of brown-adipose-tissue mitochondria 2 days after a single s.c. injection of streptozotocin (100 mg/kg). **Diabetes** caused an 80% decrease in carnitine-dependent oxidation of palmitoyl-CoA and a 50-60% decrease in overt carnitine palmitoyltransferase activity. An addnl. lesion in brown-adipose-tissue mitochondrial oxidative capacity was also indicated, since **diabetes** increased by 30-50% the rate of oxidation under uncoupled conditions of several respiratory substrates. This decrease in mitochondrial function was accompanied by an .apprx.30% decrease in the abundance of cytochromes (α + α3) and total cytochromes b.

IT **1763-10-6**, Palmitoyl-CoA
RL: RCT (Reactant); RACT (Reactant or reagent)
(oxidation of, carnitine-dependent, in brown adipose tissue mitochondria in **diabetes** mellitus)

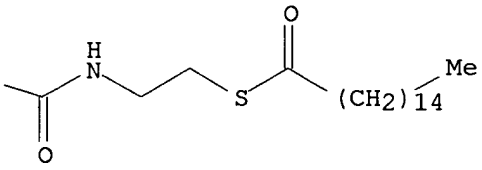
RN 1763-10-6 CAPLUS
CN Coenzyme A, S-hexadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



L9 ANSWER 16 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN
1988:92242 Document No. 108:92242 Use of a selectively permeabilized isolated rat hepatocyte preparation to study changes in the properties of overt carnitine palmitoyltransferase activity in situ. Boon, Mark R.;

Zammit, Victor A. (Hannah Res. Inst., Ayr, KA6 5HL, UK). Biochemical Journal, 249(3), 645-52 (English) 1988. CODEN: BIJOAK. ISSN: 0306-3275.

AB A permeabilized isolated rat liver cell preparation was developed to achieve selective permeabilization of the cell membrane to metabolites and to allow the assay of mitochondrial overt carnitine palmitoyltransferase I (CPT I) activity in situ. By performing the digitonin-induced permeabilization in the presence of F- and divalent-metal-cation sequestrants, it was possible to demonstrate that the activity of other enzymes which are regulated by reversible phosphorylation was preserved during the procedure and subsequent washing of cells before assay. CPT activity at a suboptimal palmitoyl-CoA concentration was almost totally (.apprx.90%) inhibited by malonyl-CoA, indicating that mitochondrial CPT I was largely measured in this preparation The palmitoyl-CoA-saturation and malonyl-CoA-inhibition curves for CPT activity in permeabilized cells were very similar to those obtained previously for the enzyme in isolated liver mitochondria. Moreover, starvation and **diabetes** had the same effects on enzyme activity, affinity for palmitoyl-CoA, and malonyl-CoA sensitivity of CPT I in isolated cells as found in isolated mitochondria. These physiol. induced changes persisted through the cell preparation and incubation period. Neither incubation of cells with glucagon or insulin nor incubation with pyruvate and lactate before permeabilization resulted in alterations of these parameters of CPT I in isolated cells. The results are discussed in relation to the temporal relationships of changes in the activity and properties of CPT I in vivo in relation to the effects of insulin and glucagon on fatty acid metabolism in vivo.

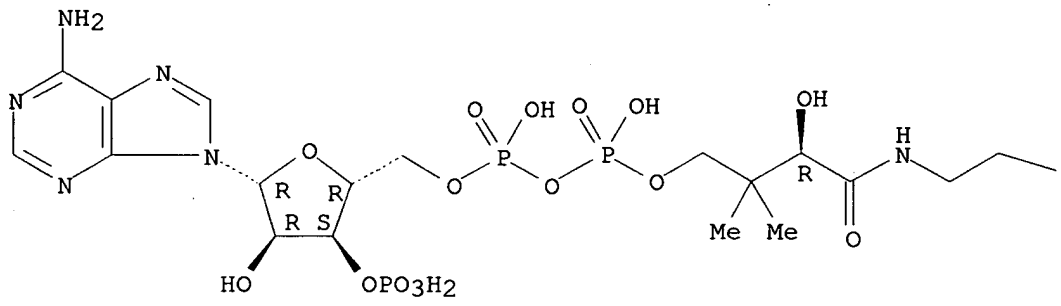
IT **1763-10-6**, Palmitoyl-CoA
RL: BIOL (Biological study)
(carnitine palmitoyltransferase I regulation by, in permeabilized hepatocytes)

RN 1763-10-6 CAPLUS

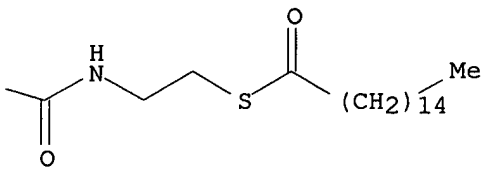
CN Coenzyme A, S-hexadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



L9 ANSWER 17 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN
1988:73133 Document No. 108:73133 Role of carnitine palmitoyltransferase I in the regulation of hepatic ketogenesis during the onset and reversal of chronic **diabetes**. Grantham, Barbara D.; Zammit, Victor A. (Hannah Res. Inst., Ayr, KA6 5HL, UK). Biochemical Journal, 249(2), 409-14 (English) 1988. CODEN: BIJOAK. ISSN: 0306-3275.

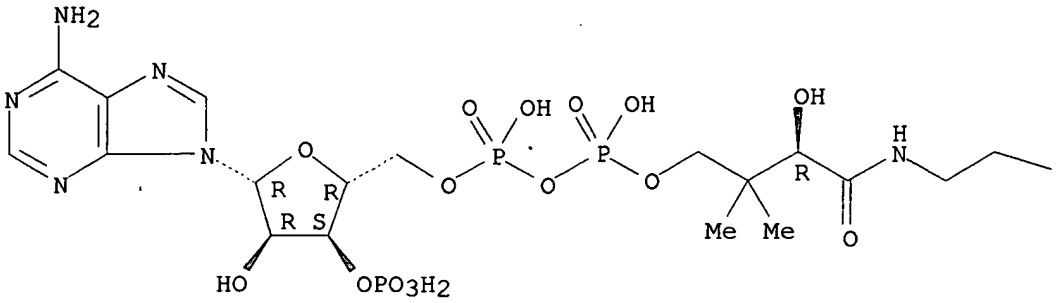
AB The kinetic properties. of overt carnitine palmitoyltransferase (CPT I, EC 2.3.1.21) were studied in rat liver mitochondria isolated from untreated, diabetic, and insulin-treated diabetic animals. The development of hyperketonemia over the first 5 days of insulin withdrawal from streptozotocin-treated rats was accompanied by parallel increases in the activity of CPT I and in the I0.5 (concentration required to produce 50% inhibition) of the enzyme for malonyl-CoA. The rapid reversal of the ketotic state by treatment of chronically diabetic rats with 6 units of regular insulin was not accompanied by any change in the properties of CPT I over the first 4 h. Higher doses of insulin (15 units), delivered throughout a 4 h period, resulted in an increase in the affinity of CPT I for malonyl-CoA, but the sensitivity of the enzyme to the inhibitor was still lower than in mitochondria from normal animals. Conversely, when insulin treatment was continued over a 24 h period, full restoration of the sensitivity of the enzyme to malonyl-CoA was achieved. However, the activity of the enzyme was only decreased marginally. These results are discussed in terms of the possibility that the major regulatory sites of the rate of hepatic oxidation may vary in different phases of the induction and reversal of chronic **diabetes**.

IT **1763-10-6**, Palmitoyl-CoA
RL: RCT (Reactant); RACT (Reactant or reagent)
(reaction of, with carnitine palmitoyltransferase I, in **diabetes** mellitus pathogenesis and reversal)

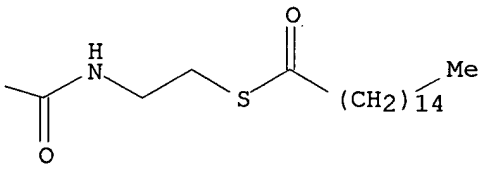
RN 1763-10-6 CAPLUS
CN Coenzyme A, S-hexadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



L9 ANSWER 18 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN
1989:108658 Document No. 110:108658 Differentiation of rapid and slower-acting effects of insulin on mitochondrial processes in brown adipose tissue from streptozotocin-diabetic rats. Gualberto, Antonio; Saggerson, E. David (Dep. Biochem., Univ. Coll. London, London, WC1E 6BT, UK). Biochemical Journal, 258(1), 309-11 (English) 1989. CODEN: BIJOAK. ISSN: 0306-3275.

AB Insulin treatment of streptozotocin-diabetic rats restores the depressed palmitoyl-group oxidation observed in brown adipose tissue mitochondria from diabetic rats. A relatively rapid effect of insulin (5 h) to increase carnitine-dependent oxidation of palmitoyl-CoA and to increase overt carnitine palmitoyltransferase activity is differentiated from a slower effect of the hormone (1 day) to increase palmitoylcarnitine oxidation

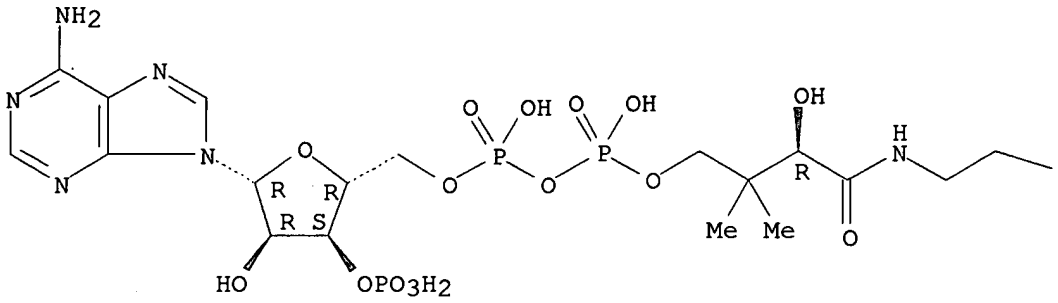
IT **1763-10-6**, Palmitoyl-CoA
RL: RCT (Reactant); RACT (Reactant or reagent)
(oxidation of, by brown adipose tissue mitochondria in **diabetes** mellitus, insulin effect on)

RN 1763-10-6 CAPLUS

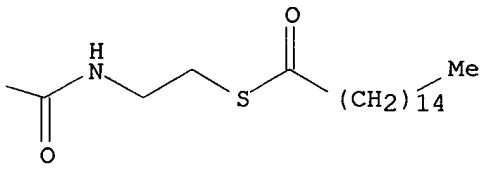
CN Coenzyme A, S-hexadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



L9 ANSWER 19 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN

1990:116668 Document No. 112:116668 Enzyme site-specific changes in hepatic microsomal fatty acid chain elongation in streptozotocin-induced diabetic rats. Suneja, Sanoj K.; Osei, Peter; Cook, Lynda; Nagi, Mahmoud N.; Cinti, Dominick L. (Health Cent., Univ. Connecticut, Farmington, CT, USA). Biochimica et Biophysica Acta, 1042(1), 81-5 (English) 1990. CODEN: BBACAQ. ISSN: 0006-3002.

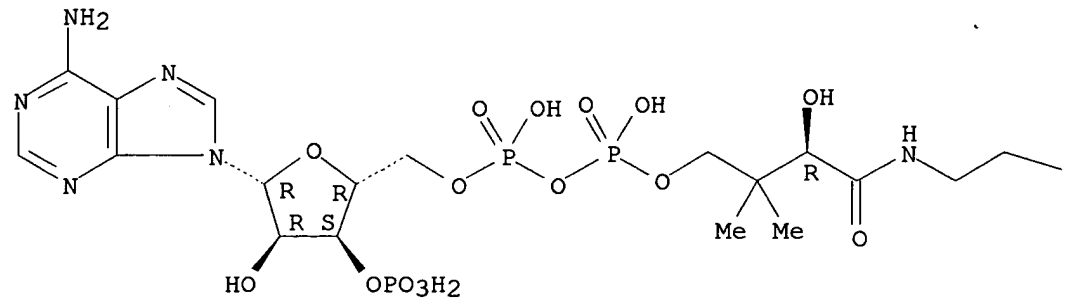
AB The hepatic microsomal fatty acid chain elongation of palmitoyl-CoA and γ-linolenoyl-CoA was diminished by 40-50% in male Sprague-Dawley rats made diabetic for 2 and 4 wk following the i.v. administration of a single dose (65 mg/kg) of streptozotocin. Anal. of the activities of the 4 enzymic components showed that only 1 enzyme, the condensing enzyme, which catalyzes the initial and rate-limiting step in chain elongation, was altered by the diabetic state. Both chain elongation and condensation activities were depressed to the same extent, whereas β-ketoacyl-CoA reductase, β-hydroxyacyl-CoA dehydrase and trans-2-enoyl-CoA reductase activities were the same as the values obtained with nondiabetic controls. Two-week administration of 10 units of insulin per day to rats which were diabetic for a 2-wk period resulted in the reversal of the reduced palmitoyl-CoA elongation and condensation activities to control values. However, neither the condensation nor the elongation of γ-linolenoyl-CoA was reversed by the insulin treatment. These results support the notion of multiple condensing enzymes or chain elongation systems.

IT **1763-10-6**, Palmitoyl-CoA **27843-61-4**, γ-Linolenoyl-CoA
RL: BIOL (Biological study)
(elongation of, in liver microsomes, defect of, in **diabetes** mellitus, insulin effect on)

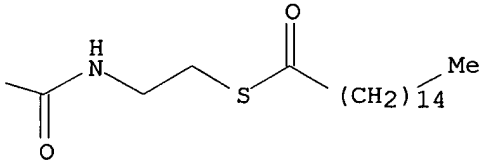
RN 1763-10-6 CAPLUS
CN Coenzyme A, S-hexadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



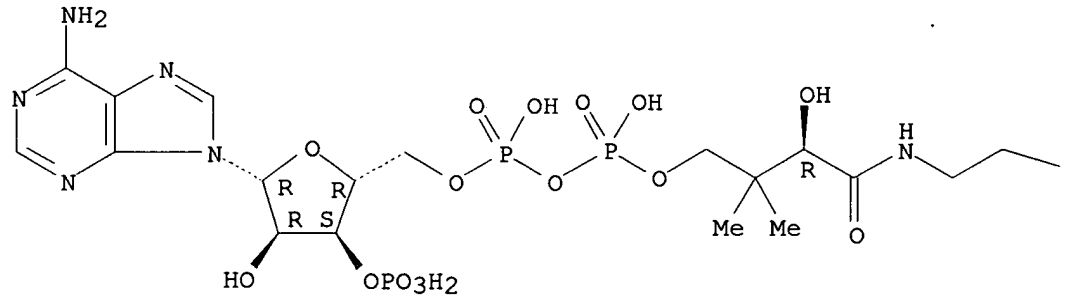
PAGE 1-B



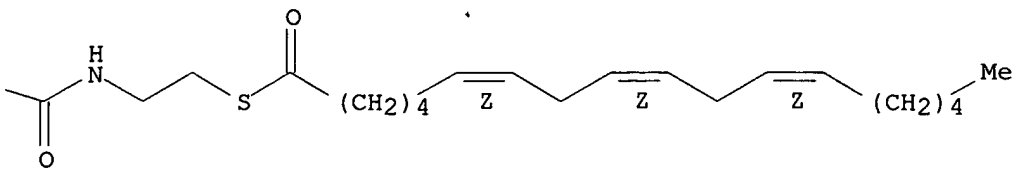
RN 27843-61-4 CAPLUS
CN Coenzyme A, S-(6Z,9Z,12Z)-6,9,12-octadecatrienoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

PAGE 1-A



PAGE 1-B



L9 ANSWER 20 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN
1992:103672 Document No. 116:103672 Evidence for insulin dependent hepatic microsomal γ-linolenic acid chain elongation in spontaneously diabetic Wistar BB rats. Mimouni, Virginie; Narce, Michel; Poisson, Jean Pierre (Fac. Sci., Univ. Bourgogne, Dijon, 21004, Fr.). Biochimica et Biophysica Acta, 1133(2), 187-92 (English) 1992. CODEN: BBACAQ. ISSN: 0006-3002.

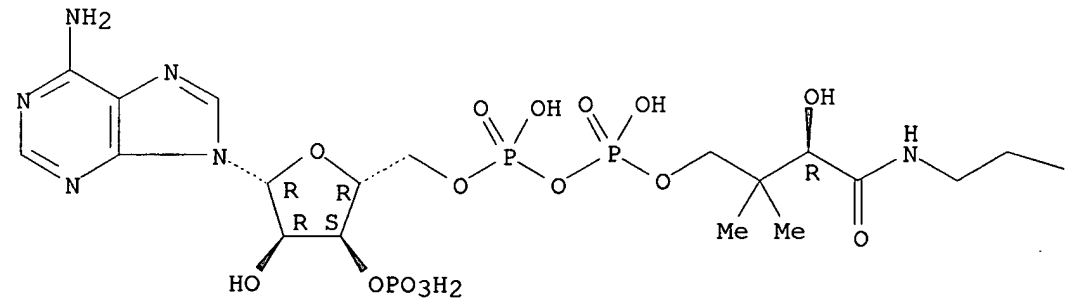
AB This study investigated hepatic microsomal γ-linolenoyl-CoA elongation and fatty acid composition of liver microsomes in spontaneously diabetic Wistar BB rats. The liver microsomal γ-linolenoyl-CoA elongation was decreased in diabetic Wistar BB rats during both normo- and hyperglycemic periods and restored during the hypoglycemic period following insulin treatment. These results are in agreement with previously reported data on linoleic acid Δ6 and Δ5 desatns. and support the non-parallel relationship between the chain elongation system and the glycemia. The fatty acid composition of BB rat liver microsomes was only partially consistent with the γ-linolenoyl-CoA elongation activity at the different periods of glycemia, probably because factors other than elongation impairments were involved in the evolution of fatty acid composition

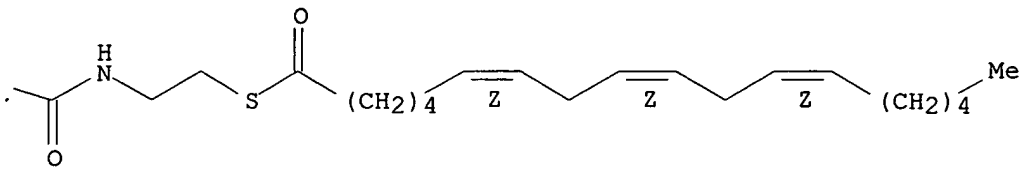
IT **27843-61-4**, γ-Linolenoyl-CoA
RL: PROC (Process)
(elongation of, during fatty acid formation, in liver microsomes, insulin dependency of, in **diabetes** mellitus)

RN 27843-61-4 CAPLUS
CN Coenzyme A, S-(6Z,9Z,12Z)-6,9,12-octadecatrienoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

PAGE 1-A





=> d 21-40 cbib abs hitstr

L9 ANSWER 21 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN
1993:536747 Document No. 119:136747 Increased platelet aggregation and fatty acid oxidation in diabetic rats. Iida, Naohiro; Iida, Reimi; Takeyama, Naoshi; Tanaka, Takaya (Dep. Emerg. Crit. Care Med., Kansai Med. Univ., Moriguchi, 570, Japan). Biochemistry and Molecular Biology International, 30(1), 177-85 (English) 1993. CODEN: BMBIES. ISSN: 1039-9712.

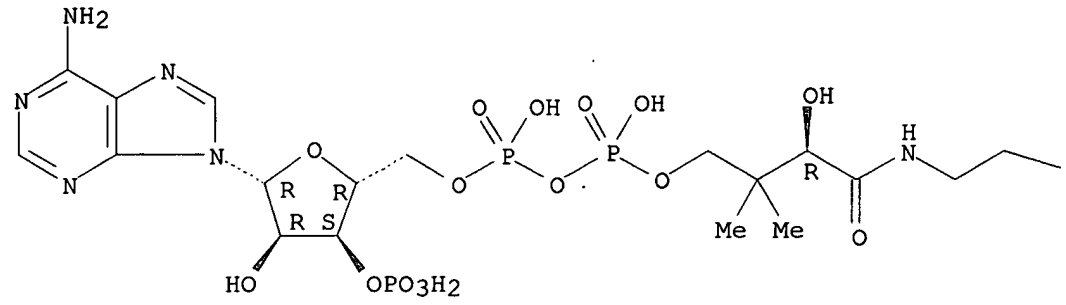
AB The effects of **diabetes** on fatty acid oxidation in platelets was determined in streptozotocin-diabetic rats. In platelets isolated from the diabetic rats, the O consumption which reflects mainly the degree of fatty acid oxidation and ADP- and thrombin-induced aggregation were increased as compared to nondiabetic rat platelets. Carnitine palmitoyltransferase I (CPT I), the rate-limiting enzyme for fatty acid oxidation, in platelets obtained from diabetic rats showed a higher Vmax for palmitoyl-CoA and an increased I50 (concentration giving 50% inhibition of CPT I activity) for malonyl-CoA inhibition. These changes observed in fatty acid oxidation in platelets derived from **diabetes** returned to the control levels after insulin therapy. When platelets were stimulated with thrombin, platelet CPT I activity increased over time in both diabetic and nondiabetic rats. Thus, fatty acid oxidation in platelets, as in the liver, probably is regulated by insulin and both increased CPT I activity and decreased sensitivity to malonyl-CoA inhibition are attributable to enhanced platelet fatty acid oxidation in diabetic rats.

IT **1763-10-6**, Palmitoyl CoA
RL: BIOL (Biological study)
(carnitine palmitoyltransferase I inhibition by, in blood platelets in **diabetes** mellitus, insulin effect on, fatty acid oxidation in relation to)

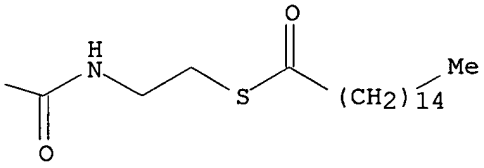
RN 1763-10-6 CAPLUS
CN Coenzyme A, S-hexadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



L9 ANSWER 22 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN

1996:388439 Document No. 125:52163 Fatty acids and anionic phospholipids alter the palmitoylcoenzyme A kinetics of hepatic monoacylglycerol acyltransferase in Triton X-100 mixed micelles. Coleman, Rosalind A.; Wang, P.; Bhat, B. Ganesh (Department of Nutrition, University of North Carolina, Chapel Hill, NC, 27599-7400, USA). Biochemistry, 35(29), 9576-9583 (English) 1996. CODEN: BICHAW. ISSN: 0006-2960. Publisher: American Chemical Society.

AB To gain a better understanding of the kinetics of activation and inhibition of hepatic monoacylglycerol acyltransferase (MGAT) (EC 2.3.1.22) by fatty acid, the authors examined the effect of fatty acid with respect to MGAT's long-chain acyl-CoA substrate in Triton X-100 mixed micelles. At concns. between 2.5 and 5.3 mol %, oleic acid stimulated MGAT activity 2-fold, whereas oleic acid inhibited MGAT at concns. higher than 7.5 mol %. The dependence on palmitoyl-CoA was highly cooperative with a Hill constant of greater than 2.4. When present at less than 3 mol %, oleic acid eliminated the lag in the dependence curve. When concns. of oleic acid were higher than 3 mol %, Michaelis-Menten kinetics were observed with an apparent Km value of about 54 μM for palmitoyl-CoA but with progressively decreasing Vmax values. This effect was not observed with octanoic acid, suggesting that the medium-chain fatty acid is unable to associate stably with the mixed micelle and, thus, cannot substantially alter substrate affinity. When anionic phospholipids were tested, phosphatidic acid, lysophosphatidic acid, phosphatidylserine, and phosphatidylinositol eliminated some of the lag in activation by palmitoyl-CoA. At high molar concns. of the anionic lipid activators, apparent Km values ranged from 77 μM for phosphatidic acid to 196 μM for phosphatidylinositol. Zwitterionic phospholipids had no effect, nor did the non-phospholipid activators bovine serum albumin or sn-1,2-diacylglycerol. CaCl2, but not

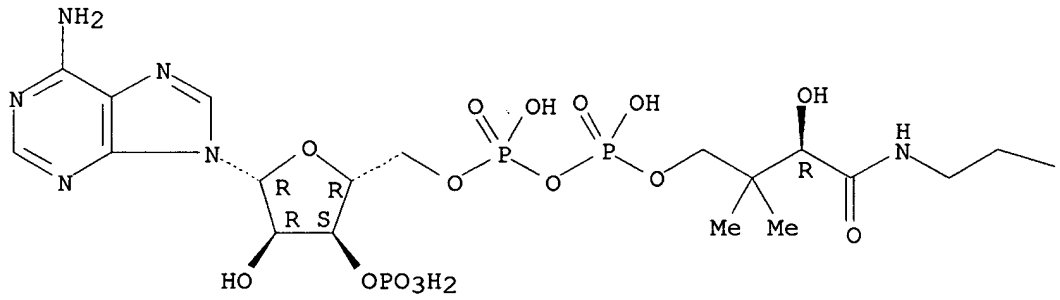
neomycin or KCl, could overcome the inhibitory effect of oleic acid; thus, the inhibitory effect of fatty acid did not appear to occur by electrostatic interactions. These blockers did not change the effects observed with the anionic phospholipid activators or with the inhibitor, sphingosine. An altered Km for palmitoyl-CoA in the presence of fatty acid or anionic phospholipid suggests that both long-chain fatty acids and phospholipid cofactors may induce a conformational change in MGAT, thereby altering the enzyme's affinity for its long-chain acyl-CoA substrate. These data further support the hypothesis that the synthesis of glycerolipids via the monoacylglycerol pathway may be highly regulated via a variety of lipid second messengers such as phosphatidic acid and diacylglycerol, as well as by the influx of fatty acids derived from high-fat diets, or from the hydrolysis of adipocyte triacylglycerol during fasting or **diabetes**.

IT **1763-10-6**, Palmitoyl-CoA
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(fatty acids and anionic phospholipids alter palmitoyl-CoA kinetics of hepatic monoacylglycerol acyltransferase in Triton X-100 mixed micelles)

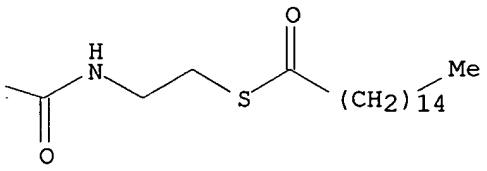
RN 1763-10-6 CAPLUS
CN Coenzyme A, S-hexadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



L9 ANSWER 23 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN
1999:16486 Document No. 130:207774 Maturation of peroxisomes in differentiating human hepatoblastoma cells (HepG2): possible involvement of the peroxisome proliferator-activated receptor α (**PPAR** α). Stier, Harald; Fahimi, H. Dariush; Van Veldhoven, Paul P.;

Mannaerts, Guy P.; Volkl, Alfred; Baumgart, Eveline (Institut fur Anatomie and Zellbiologie, Abteilung Medizinische Zellbiologie, Medizinische Fakultat, Ruprecht-Karls-Universitat Heidelberg, Heidelberg, D-69120, Germany). Differentiation (Berlin), 64(1), 55-66 (English) 1998. CODEN: DFFNAW. ISSN: 0301-4681. Publisher: Springer-Verlag.

AB We have studied the alterations of peroxisomes in the human hepatoblastoma cell line HepG2, induced to differentiate by long-term cultivation (20 days without passaging) using morphol. and biochem. techniques as well as mRNA anal. Ultrastructural studies revealed alterations in shape and size of peroxisomes, with significant increases in mean diameter and formation of small clusters exhibiting heterogeneous staining for catalase after 20 days in culture. These alterations of peroxisomes correspond to the changes described during the maturation process from prenatal to adult human hepatocytes. As revealed by Northern and Western blotting there was marked elevation of the mRNA (190%) and protein (180%) of the peroxisomal branched-chain acyl-CoA oxidase. This protein is the key regulatory enzyme for the side chain oxidation of cholesterol for bile acid synthesis, a pathway associated with mature hepatocytes. Concomitantly a marked increase of bile canaliculi was noted by light and electron microscopy. This differentiation process was confirmed also by the increase of albumin synthesis (mRNA: 160%; protein: 190%) which is generally used as a differentiation marker of hepatocytes in culture. Interestingly, the mRNA for peroxisome proliferator-activated receptor α (**PPAR** α) increased drastically by almost 390% and its corresponding protein by 150%, suggesting its involvement in maturation of the peroxisomal compartment in differentiating HepG2 cells. In contrast to the well-known increases during the drug-induced peroxisome proliferation of cytochrome P 450 4A, multifunctional enzyme 1, palmitoyl-CoA oxidase and the 70-kDa peroxisomal membrane protein, those proteins were either not altered or only slightly elevated during the differentiation process, suggesting that peroxisome proliferation and maturation are two distinct and differentially regulated processes.

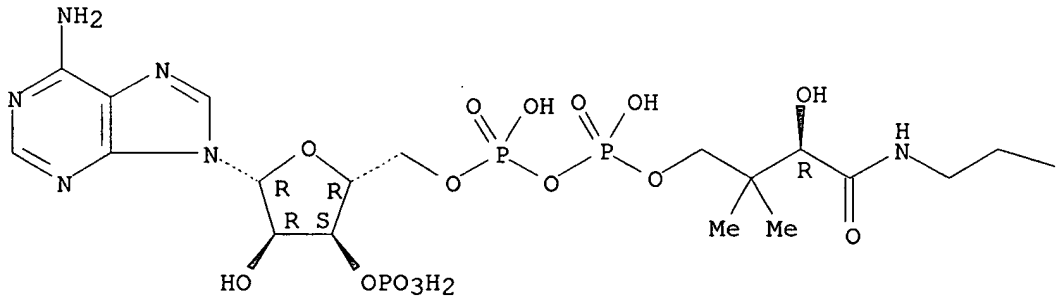
IT **1763-10-6**, Palmitoyl-CoA
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(elevation of peroxisomal branched-chain acyl-CoA oxidase and palmitoyl-CoA in differentiating human hepatoblastoma cells (HepG2))

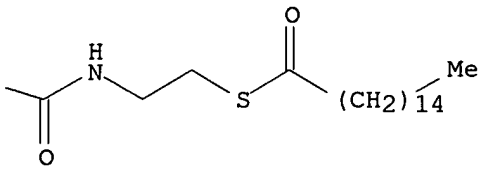
RN 1763-10-6 CAPLUS

CN Coenzyme A, S-hexadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A





L9 ANSWER 24 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN

1999:15585 Document No. 130:151378 K+ current inhibition by amphiphilic fatty acid metabolites in rat ventricular myocytes. Xu, Zhi; Rozanski, George J. (Department of Physiology and Biophysics, University of Nebraska Medical Center, Omaha, NE, 68198-4575, USA). American Journal of Physiology, 275(6, Pt. 1), C1660-C1667 (English) 1998. CODEN: AJPHAP. ISSN: 0002-9513. Publisher: American Physiological Society.

AB Fatty acid metabolites accumulate in the heart under pathophysiol. conditions that affect β-oxidation and can elicit marked electrophysiol. changes that are arrhythmogenic. The purpose of the present study was to determine the impact of amphiphilic fatty acid metabolites on K+ currents that control cardiac refractoriness and excitability. Transient outward (Ito) and inward rectifier (IK1) K+ currents were recorded by the whole cell voltage-clamp technique in rat ventricular myocytes, and the effects of two major fatty acid metabolites were examined: palmitoylcarnitine and palmitoyl-CoA (palmitoyl-CoA). Palmitoylcarnitine (0.5-10 μM) caused a concentration-dependent decrease in Ito d. in myocytes internally dialyzed with the amphiphile; 10 μM reduced mean Ito d. at +60 mV by 62% compared with control ($P < 0.05$). In contrast, external palmitoylcarnitine at the same concns. had no effect, nor did internal dialysis significantly alter IK1. Dialysis with palmitoyl-CoA (1-10 μM) produced a smaller decrease in Ito d. compared with that produced by palmitoylcarnitine; 10 μM reduced mean Ito d. at +60 mV by 37% compared with control ($P < 0.05$). Both metabolites delayed recovery of Ito from inactivation but did not affect voltage-dependent properties. Moreover, the effects of palmitoylcarnitine were relatively specific, as neither palmitate (10 μM) nor carnitine (10 μM) alone significantly influenced it when added to the pipet solution These data therefore suggest that amphiphilic fatty acid metabolites downregulate Ito channels by a mechanism confined to the cytoplasmic side of the membrane. This decrease in cardiac K+ channel activity may delay repolarization under pathophysiol. conditions in which amphiphile accumulation is postulated to occur, such as **diabetes** mellitus or myocardial infarction.

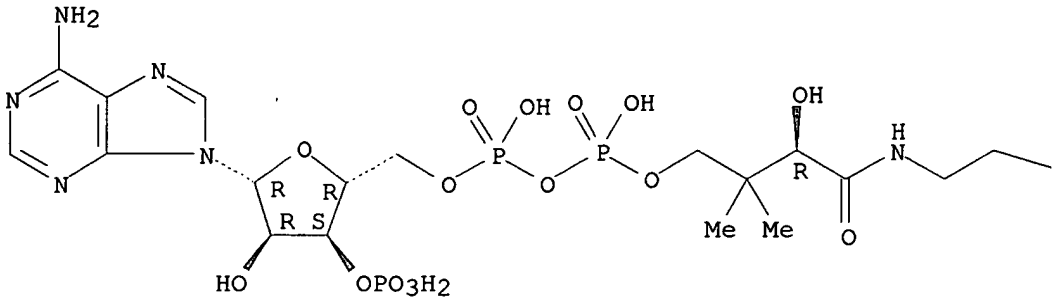
IT **1763-10-6**, Palmitoyl-CoA
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(K+ current inhibition by amphiphilic fatty acid metabolites in rat ventricular myocytes)

RN 1763-10-6 CAPLUS

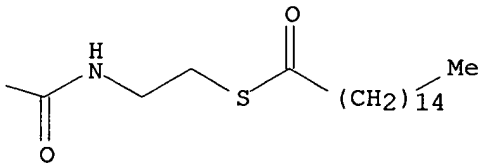
CN Coenzyme A, S-hexadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



L9 ANSWER 25 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN

1998:797445 Document No. 130:151958 Lipoapoptosis in beta-cells of obese prediabetic fa/fa rats. Role of serine palmitoyltransferase overexpression. Shimabukuro, Michio; Higa, Moritake; Zhou, Yan-Ting; Wang, May-Yun; Newgard, Christopher B.; Unger, Roger H. (Gifford Laboratories for Diabetes Research, Department of Internal Medicine, University of Texas Southwestern Medical Center, Dallas, TX, 75235, USA). Journal of Biological Chemistry, 273(49), 32487-32490 (English) 1998. CODEN: JBCHA3. ISSN: 0021-9258. Publisher: American Society for Biochemistry and Molecular Biology.

AB We reported that the lipoapoptosis of beta-cells observed in fat-laden islets of obese fa/fa Zucker Diabetic Fatty (ZDF) rats results from overprodn. of ceramide, an initiator of the apoptotic cascade and is induced by long-chain fatty acids (FA). Whereas the ceramide of cytokine-induced apoptosis may be derived from sphingomyelin hydrolysis, FA-induced ceramide overprodn. seems to be derived from FA. We therefore semiquantified mRNA of serine palmitoyltransferase (SPT), which catalyzes the first step in ceramide synthesis. It was 2-3-fold higher in fa/fa islets than in +/+ controls. [3H]Ceramide formation from [3H]serine was 2.2-4.5-fold higher in fa/fa islets. Triacsin-C, which blocks palmitoyl-CoA synthesis, and L-cycloserine, which blocks SPT activity, completely blocked [3H]ceramide formation from [3H]serine. Islets of fa/fa rats are unresponsive to the lipopenic action of leptin, which normally depletes fat and prevents FA up-regulation of SPT. To determine the role of leptin unresponsiveness in the SPT overexpression, we transferred wild type OB-Rb cDNA to their islets; now leptin completely blocked the exaggerated FA-induced increase of SPT mRNA while reducing the fat content. Beta-cell lipoapoptosis was partially prevented in vivo by treating prediabetic ZDF rats with L-cycloserine for 2 wk. Ceramide

content and DNA fragmentation both declined 40-50%. We conclude that lipoapoptosis of ZDF rats is mediated by enhanced ceramide synthesis from FA and that blockade by SPT inhibitors prevents lipoapoptosis.

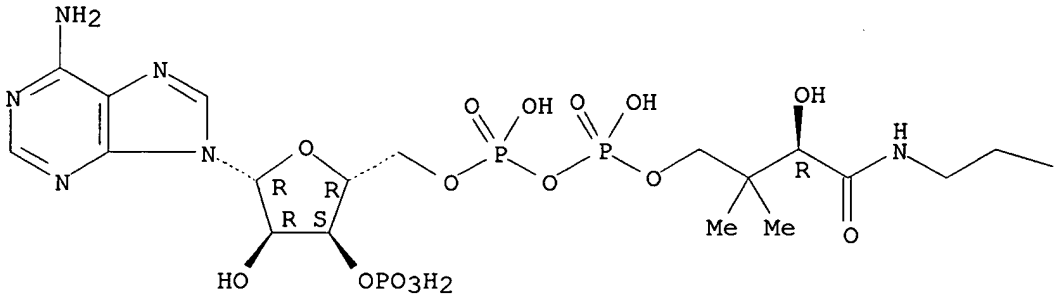
IT **1763-10-6**, Palmitoyl CoA
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(lipoapoptosis in β cells of obese prediabetic fa/fa rats is mediated by enhanced ceramide biosynthesis from serine and palmitoyl CoA by serine palmitoyltransferase mRNA overexpression upregulated by fatty acids)

RN 1763-10-6 CAPLUS

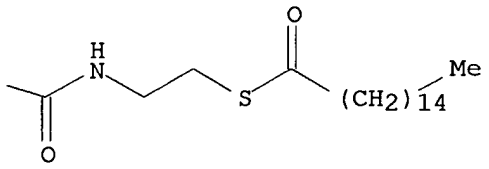
CN Coenzyme A, S-hexadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



L9 ANSWER 26 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN

1998:672045 Document No. 130:11625 Mechanism of cloned ATP-sensitive potassium channel activation by oleoyl-CoA. Gribble, Fiona M.; Proks, Peter; Corkey, Barbara E.; Ashcroft, Frances M. (University Laboratory of Physiology, Oxford, OX1 3PT, UK). Journal of Biological Chemistry, 273(41), 26383-26387 (English) 1998. CODEN: JBCHA3. ISSN: 0021-9258. Publisher: American Society for Biochemistry and Molecular Biology.

AB Insulin secretion from pancreatic beta cells is coupled to cell metabolism through closure of ATP-sensitive potassium (KATP) channels, which comprise Kir6.2 and sulfonylurea receptor (SUR1) subunits. Although metabolic regulation of KATP channel activity is believed to be mediated principally by the adenine nucleotides, other metabolic intermediates, including long chain acyl-CoA esters, may also be involved. We recorded macroscopic and single-channel currents from Xenopus oocytes expressing either Kir6.2/SUR1 or Kir6.2ΔC36 (which forms channels in the absence of SUR1).

Oleoyl-CoA (1 μM) activated both wild-type Kir6.2/SUR1 and Kir6.2ΔC36 macroscopic currents, .apprx.2-fold, by increasing the number and open probability of Kir6.2/SUR1 and Kir6.2ΔC36 channels. It was ineffective on the related Kir subunit Kir1.1a. Oleoyl-CoA also impaired channel inhibition by ATP, increasing the Ki values for both Kir6.2/SUR1 and Kir6.2ΔC36 currents by .apprx.3-fold. Our results indicate that activation of KATP channels by oleoyl-CoA results from an interaction with the Kir6.2 subunit, unlike the stimulatory effects of MgADP and diazoxide which are mediated through SUR1. The increased activity and reduced ATP sensitivity of KATP channels by oleoyl-CoA might contribute of the impaired insulin secretion observed in non-insulin-dependent **diabetes** mellitus.

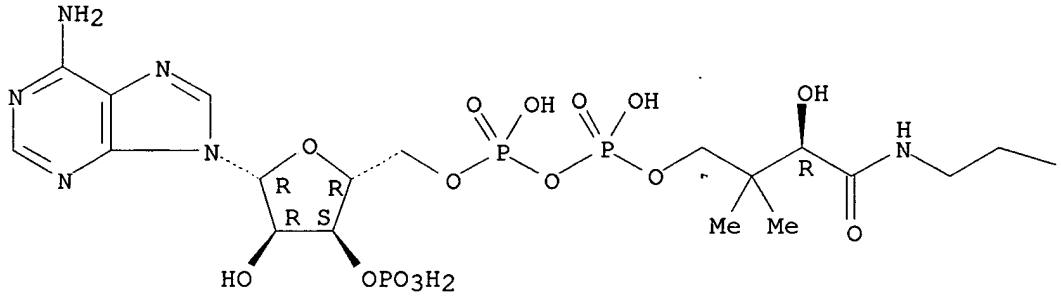
IT **1716-06-9**, Oleoyl-CoA
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(ATP-sensitive potassium channel activation by oleoyl-CoA and mechanism therefor)

RN 1716-06-9 CAPLUS

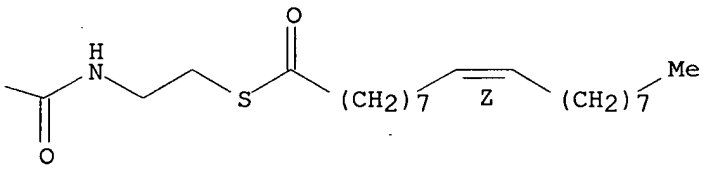
CN Coenzyme A, S-(9Z)-9-octadecenoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

PAGE 1-A



PAGE 1-B



L9 ANSWER 27 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN

1998:253636 Document No. 129:37126 Fatty acyl-CoA thioesters are ligands of hepatic nuclear factor-4α. Hertz, Rachel; Magenheim, Judith; Berman, Inna; Bar-Tana, Jacob (Dep. of Human Nutr. and Metab., Fac. of Med., Hebrew Univ., Jerusalem, Israel). Nature (London), 392(6675), 512-516 (English) 1998. CODEN: NATUAS. ISSN: 0028-0836. Publisher: Macmillan Magazines.

AB Dietary fatty acids specifically modulate the onset and progression of various diseases, including cancer, atherogenesis, hyperlipidemia, insulin resistance and hypertension, as well as blood coagulability and fibrinolytic defects; their effects depend on their chain length and degree of saturation Hepatocyte nuclear factor-4α (HNF-4α) is an orphan transcription factor of the superfamily of nuclear receptors and controls the expression of genes that govern the pathogenesis and course of some of these diseases. Here the authors show that long-chain fatty acids directly modulate the transcriptional activity of HNF-4α by binding as their acyl-CoA thioesters to the ligand-binding domain of HNF-4α. This binding may shift the oligomeric-dimeric equilibrium of HNF-4α or may modulate the affinity of HNF-4α for its cognate promoter element, resulting in either activation or inhibition of HNF-4α transcriptional activity as a function of chain length and the degree of saturation of the fatty acyl-CoA ligands. In addition to their roles as substrates to yield energy, as an energy store, or as constituents of membrane phospholipids, dietary fatty acids may affect the course of a disease by modulating the expression of HNF-4α-controlled genes.

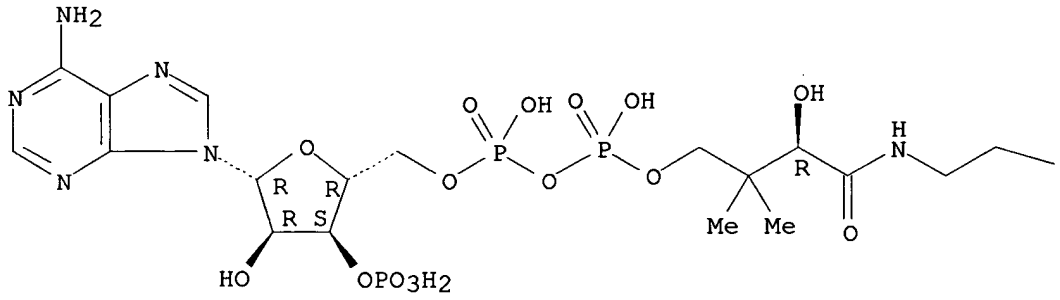
IT **362-66-3**, Stearoyl-CoA **1716-06-9**, Oleoyl-CoA
1763-10-6, Palmitoyl-CoA **3130-72-1**, Myristoyl-CoA
6709-57-5, Linoleoyl-CoA **13673-87-5**, Linolenoyl-CoA
28465-44-3 28465-45-4
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(binding of fatty acyl-CoA thioesters to hepatic nuclear factor-4α (HNF-4α), regulation of HNF-4α oligomer-dimer equilibrium by acyl-CoA thioesters, and regulation of HNF-4α transcriptional activity by fatty acids)

RN 362-66-3 CAPLUS

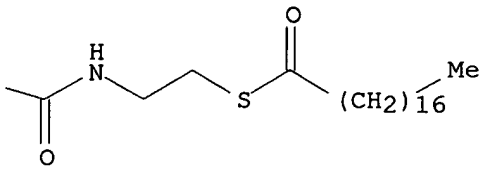
CN Coenzyme A, S-octadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



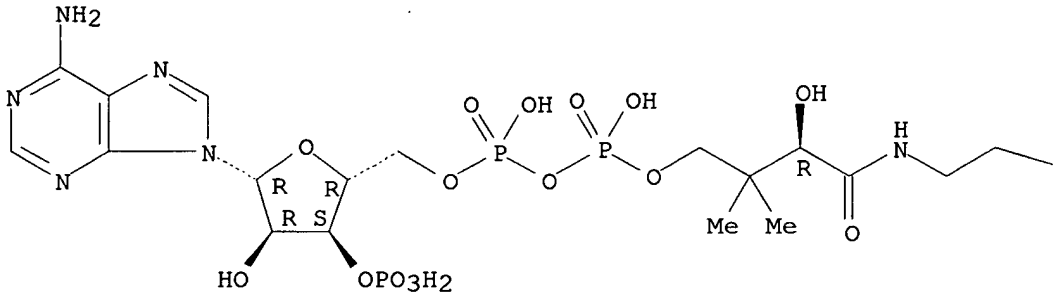
PAGE 1-B



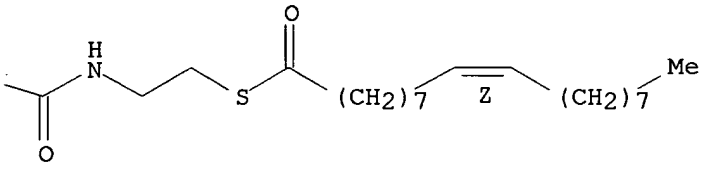
RN 1716-06-9 CAPLUS
CN Coenzyme A, S-(9Z)-9-octadecenoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

PAGE 1-A



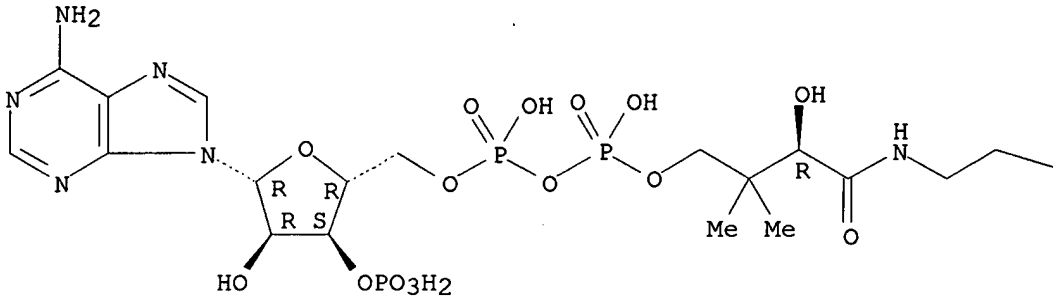
PAGE 1-B



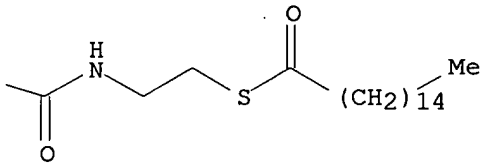
RN 1763-10-6 CAPLUS
CN Coenzyme A, S-hexadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



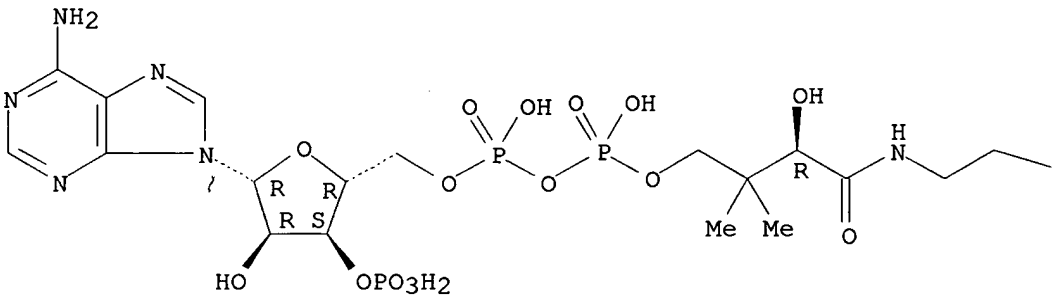
PAGE 1-B



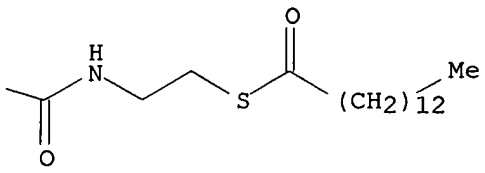
RN 3130-72-1 CAPLUS
CN Coenzyme A, S-tetradecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



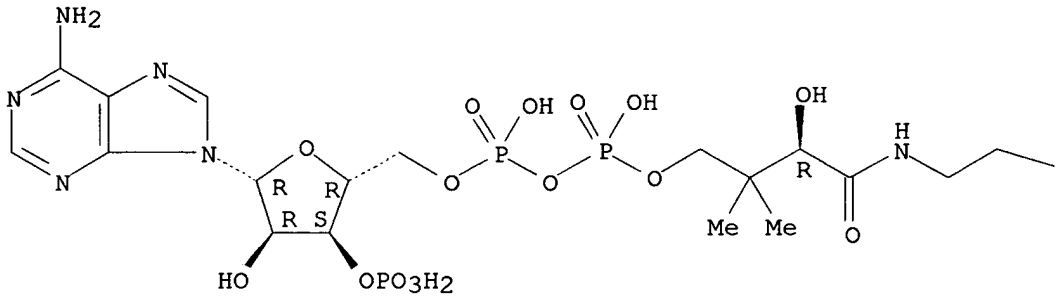
PAGE 1-B



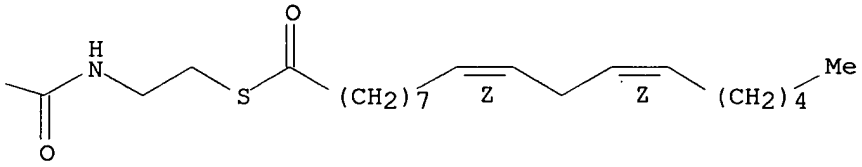
RN 6709-57-5 CAPLUS
CN Coenzyme A, S-(9Z,12Z)-9,12-octadecadienoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

PAGE 1-A

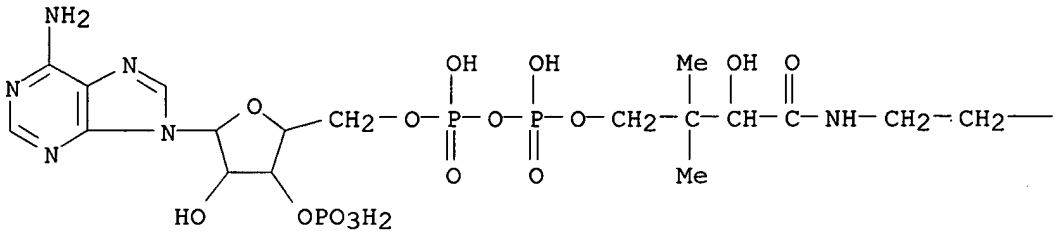


PAGE 1-B

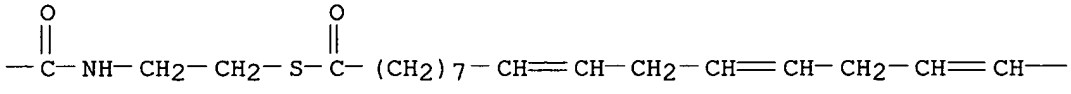


RN 13673-87-5 CAPLUS
CN Coenzyme A, S-(9Z,12Z,15Z)-9,12,15-octadecatrienoate (9CI) (CA INDEX NAME)

PAGE 1-A



PAGE 1-B



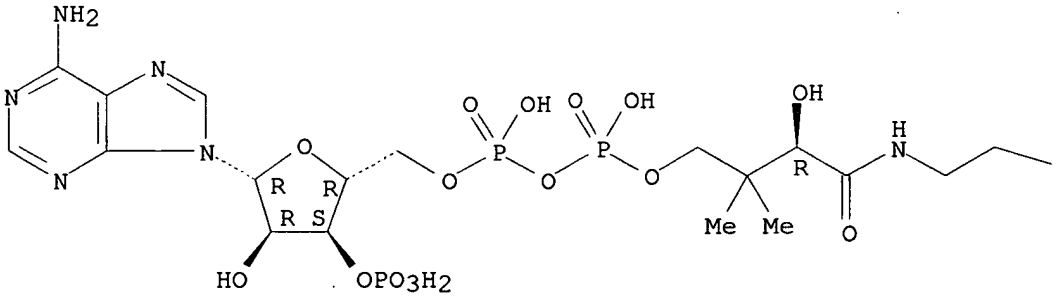
PAGE 1-C

— Et

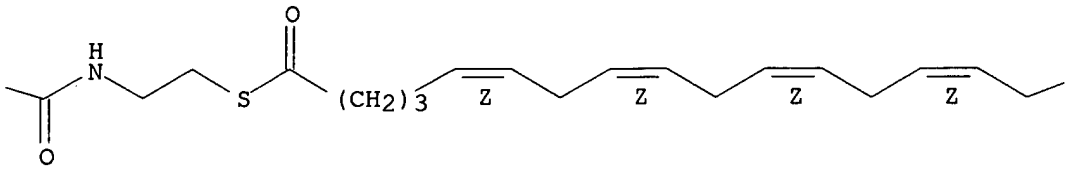
RN 28465-44-3 CAPLUS
CN Coenzyme A, S-(5Z,8Z,11Z,14Z,17Z)-5,8,11,14,17-eicosapentaenoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

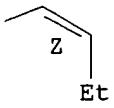
PAGE 1-A



PAGE 1-B



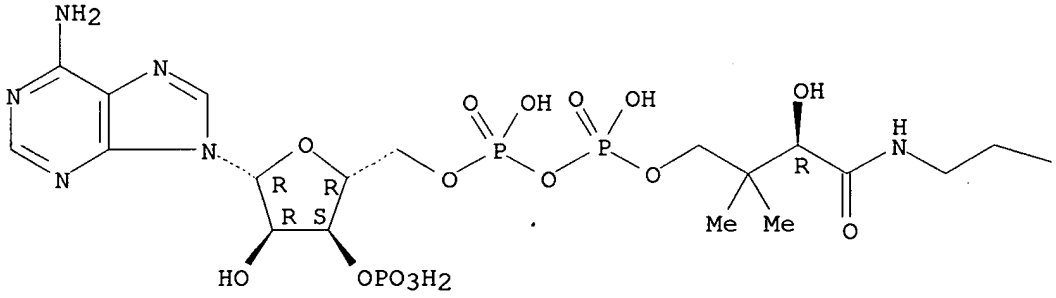
PAGE 1-C



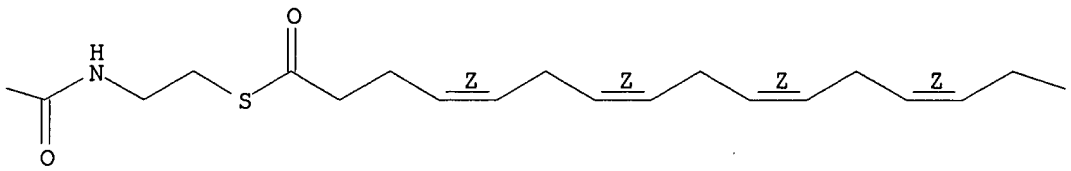
RN 28465-45-4 CAPLUS
CN Coenzyme A, S-(4Z,7Z,10Z,13Z,16Z,19Z)-4,7,10,13,16,19-docosahexaenoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

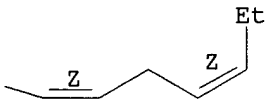
PAGE 1-A



PAGE 1-B



PAGE 1-C



L9 ANSWER 28 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN
1999:486508 Document No. 131:241393 Characterization of glucokinase mutations associated with maturity-onset **diabetes** of the young type 2 (MODY-2): different glucokinase defects lead to a common phenotype. Miller, Stephen P.; Anand, Gulshan R.; Karschnia, Elizabeth J.; Bell, Graeme I.; LaPorte, David C.; Lange, Alex J. (Department of Biochemistry, Molecular Biology and Biophysics, University of Minnesota, Minneapolis, MN, 55455, USA). Diabetes, 48(8), 1645-1651 (English) 1999. CODEN: DIAEAZ. ISSN: 0012-1797. Publisher: American Diabetes Association.
AB Glucokinase (GK) is expressed in the pancreatic β-cells and liver, and plays a key role in the regulation of glucose homeostasis. The enzymic activity and thermal stability of wild-type (WT) GK and several mutant forms associated with maturity-onset **diabetes** of the young type 2 (MODY-2) were determined by a steady-state kinetic anal. of the purified expressed proteins. The eight MODY-2 mutations studied were Ala53Ser, Val367Met, Gly80Ala, Thr168Pro, Arg36Trp, Thr209Met, Cys213Arg, and Val226Met. These missense mutations were shown to have variable effects on GK kinetic activity. The Gly80Ala and Thr168Pro mutations resulted in a large decrease in Vmax and a complete loss of the cooperative behavior associated with glucose binding. In addition, the Gly80Ala mutation resulted in a sixfold increase in the half-saturating substrate concentration (S0.5) for ATP, and Thr168Pro resulted in eight- and sixfold increases in the S0.5 values for ATP and glucose, resp. The Thr209Met and Val226Met mutations exhibited three- and fivefold increases, resp., in the S0.5 for ATP, whereas the Cys213Arg mutation resulted in a fivefold increase in the S0.5 for glucose. These mutations also led to a small yet significant reduction in Vmax. Of all the mutations studied, only the Cys213Arg mutation had reduced enzymic activity and decreased thermal stability. Two mutants, Ala53Ser and Val367Met, showed kinetic and thermal stability properties

similar to those of WT. These mutants had increased sensitivities to the known neg. effectors of GK activity, palmitoyl-CoA, and GK regulatory protein. Taken together, these results illustrate that the MODY-2 phenotype may be linked not only to kinetic alterations but also to the regulation of GK activity.

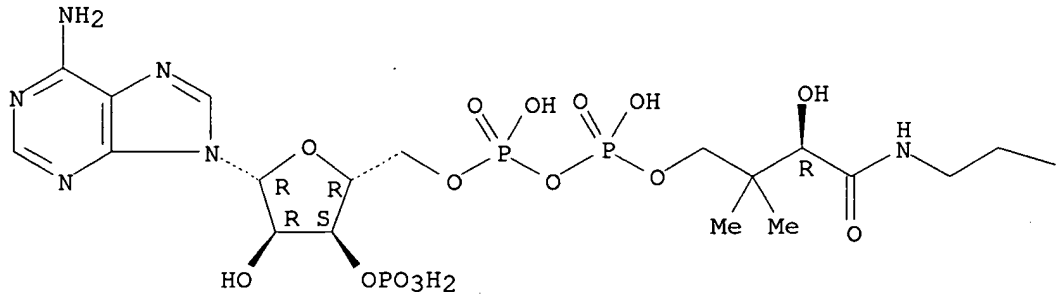
IT **1763-10-6**, Palmitoyl-CoA
RL: ADV (Adverse effect, including toxicity); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(glucokinase mutations associated with human maturity-onset **diabetes** of young type 2 (MODY-2) have different effects on enzymic kinetics, interactions with substrates/effectors, and stability but lead to common phenotype)

RN 1763-10-6 CAPLUS

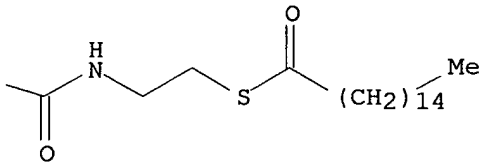
CN Coenzyme A, S-hexadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



L9 ANSWER 29 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN

2000:234876 Document No. 132:330168 Acute stimulation with long chain acyl-CoA enhances exocytosis in insulin-secreting cells (HIT T-15 and NMRI β-cells). Deeney, Jude T.; Gromada, Jesper; Hoy, Marianne; Olsen, Hervor L.; Rhodes, Christopher J.; Prentki, Marc; Berggren, Per-Olof; Corkey, Barbara E. (Rolf Luft Center for Diabetes Research, Department of Molecular Medicine, Karolinska Institutet, Stockholm, S-171 76, Swed.). Journal of Biological Chemistry, 275(13), 9363-9368 (English) 2000. CODEN: JBCHA3. ISSN: 0021-9258. Publisher: American Society for Biochemistry and Molecular Biology.

AB Non-insulin-dependent **diabetes** mellitus is associated with, in addition to impaired insulin release, elevated levels of free fatty acids

(FFA) in the blood. Insulin release is stimulated when β-cells are acutely exposed to FFA, whereas chronic exposure may inhibit glucose-induced insulin secretion. In the present study we investigated the direct effects of long chain acyl-CoA (LC-CoA), the active intracellular form of FFA, on insulin exocytosis. Palmitoyl-CoA stimulated both insulin release from streptolysin-O-permeabilized HIT cells and fusion of secretory granules to the plasma membrane of mouse pancreatic β-cells, as measured by cell capacitance. The LC-CoA effect was chain length-dependent, requiring chain lengths of at least 14 carbons. LC-CoA needed to be present to stimulate insulin release, and consequently there was no effect following its removal. The stimulatory effect was observed after inhibition of protein kinase activity and in the absence of ATP, even though both kinases and ATP, themselves, modulate exocytosis. The effect of LC-CoA was inhibited by cerulenin, which has been shown to block protein acylation. The data suggest that altered LC-CoA levels, resulting from FFA or glucose metabolism, may act directly on the exocytotic machinery to stimulate insulin release by a mechanism involving LC-CoA protein binding.

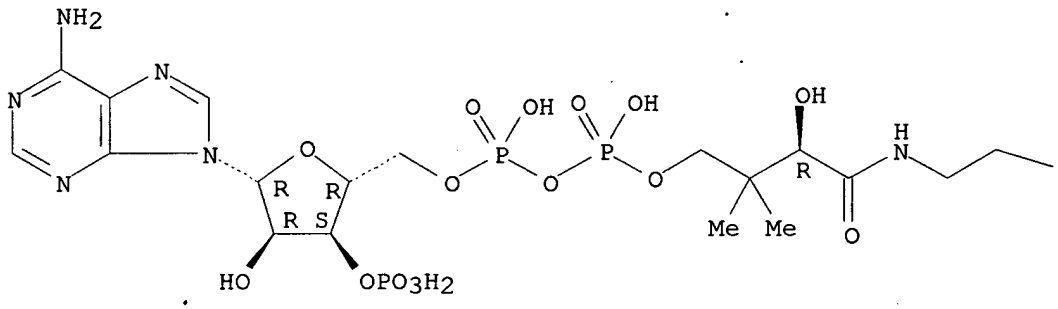
IT **1716-06-9**, Oleoyl-CoA **1763-10-6**, Palmitoyl-CoA
3130-72-1, Myristoyl-CoA **5060-32-2**, Hexanoyl-CoA
17046-56-9, Arachidonyl-CoA
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(long-chain acyl-CoA acute stimulation enhances insulin exocytosis in insulin-secreting cells and mechanism thereof)

RN 1716-06-9 CAPLUS

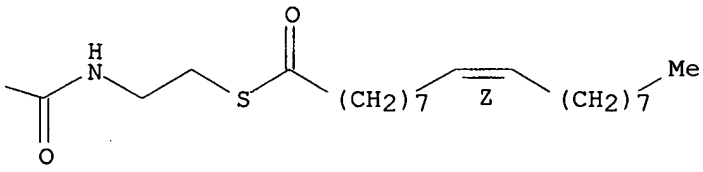
CN Coenzyme A, S-(9Z)-9-octadecenoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

PAGE 1-A



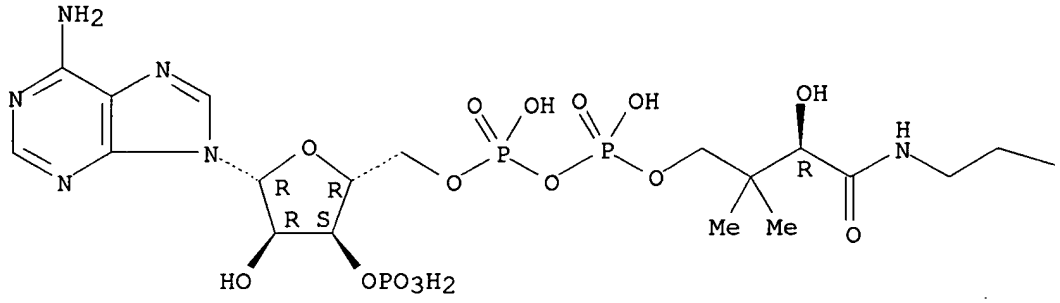
PAGE 1-B



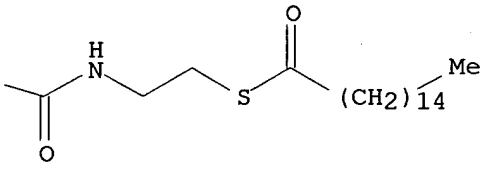
RN 1763-10-6 CAPLUS
CN Coenzyme A, S-hexadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



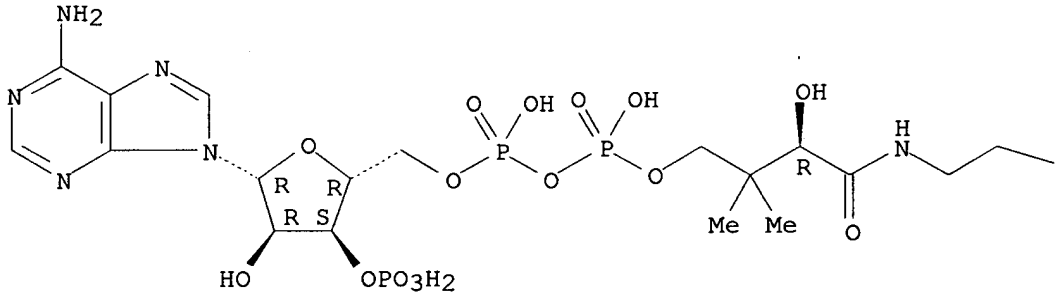
PAGE 1-B



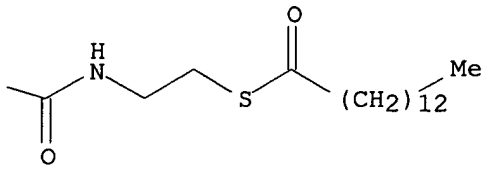
RN 3130-72-1 CAPLUS
CN Coenzyme A, S-tetradecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



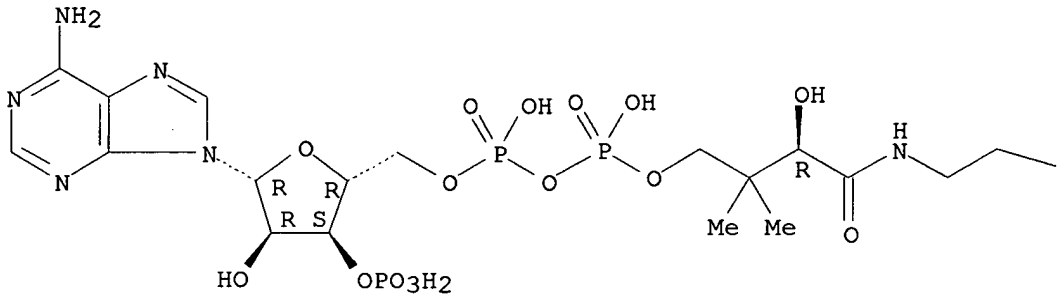
PAGE 1-B



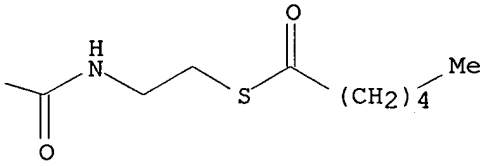
RN 5060-32-2 CAPLUS
CN Coenzyme A, S-hexanoate (7CI, 8CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



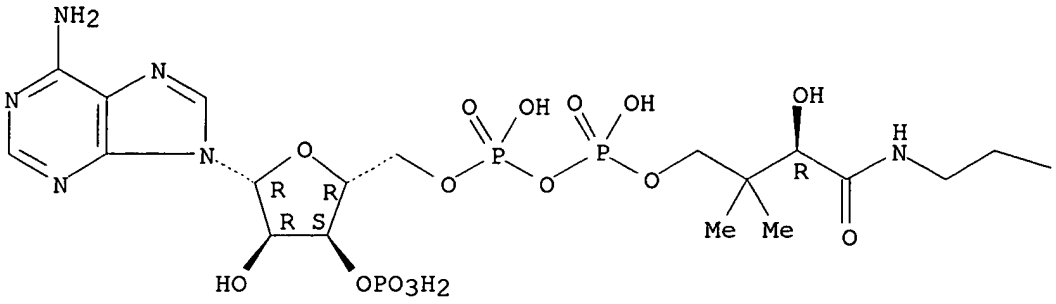
PAGE 1-B



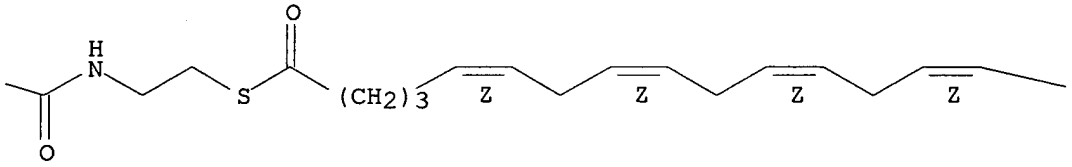
RN 17046-56-9 CAPLUS
CN Coenzyme A, S-(5Z,8Z,11Z,14Z)-5,8,11,14-eicosatetraenoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

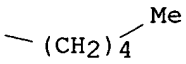
PAGE 1-A



PAGE 1-B



PAGE 1-C



L9 ANSWER 30 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN

2002:18061 Document No. 136:244813 A novel pathway for lipid biosynthesis: the direct acylation of glycerol. Lee, Douglas P.; Deonarine, Andrew S.; Kienetz, Martin; Zhu, Quansheng; Skrzypczak, Monika; Chan, Monroe; Choy, Patrick C. (Department of Biochemistry and Medical Genetics, Faculty of Medicine, University of Manitoba, Winnipeg, MB, R3E 0W3, Can.). Journal of Lipid Research, 42(12), 1979-1986 (English) 2001. CODEN: JLPRAW. ISSN: 0022-2275. Publisher: Lipid Research, Inc..

AB The acylation of glycerol-3-phosphate by acyl-CoA is regarded as the first committed step for the synthesis of the lipoidal moiety in glycerolipids. The direct acylation of glycerol in mammalian tissues has not been demonstrated. In this study, lipid biosynthesis in myoblasts and hepatocytes was reassessed by conducting pulse-chase expts. with [1,3-3H]glycerol. The results demonstrated that a portion of labeled glycerol was directly acylated to form monoacylglycerol and, subsequently, diacylglycerol and triacylglycerol. The direct acylation of glycerol

became more prominent when the glycerol-3-phosphate pathway was attenuated or when exogenous glycerol levels became elevated, Glycerol:acyl-CoA acyltransferase activity, which is responsible for the direct acylation of glycerol, was detected in the microsomal fraction of heart, liver, kidney, skeletal muscle, and brain tissues. The enzyme from pig heart microsomes displayed optimal activity at pH 6.0 and the preference for arachidonyl-CoA as the acyl donor. The apparent Km values for glycerol and arachidonyl-CoA were 1.1 mM and 0.17 mM, resp. The present study demonstrates the existence of a novel lipid biosynthetic pathway that may be important during hyperglycerolemia produced in **diabetes** or other pathol. conditions.

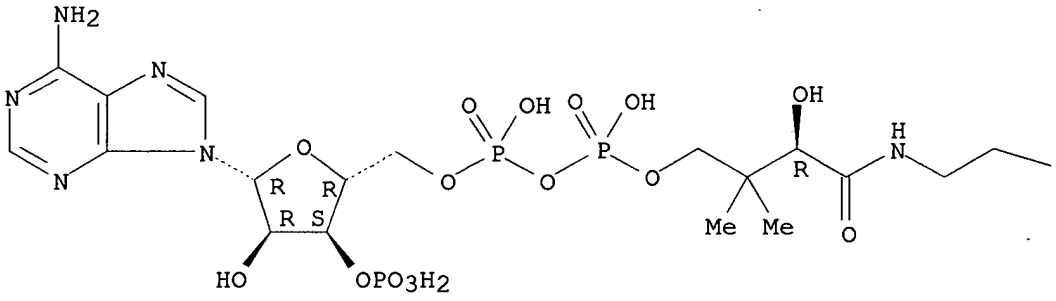
IT **17046-56-9**, Arachidonyl-CoA
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(direct acylation of glycerol in lipid biosynthesis)

RN 17046-56-9 CAPLUS

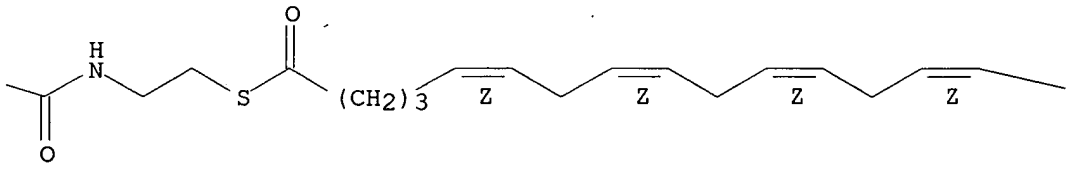
CN Coenzyme A, S-(5Z,8Z,11Z,14Z)-5,8,11,14-eicosatetraenoate (9CI) (CA INDEX NAME)

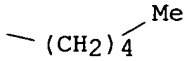
Absolute stereochemistry.
Double bond geometry as shown.

PAGE 1-A



PAGE 1-B





L9 ANSWER 31 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN

2001:560954 Document No. 135:255049 Saturated FFAs, palmitic acid and stearic acid, induce apoptosis in human granulosa cells. Mu, Yi-Ming; Yanase, Toshihiko; Nishi, Yoshihiro; Tanaka, Atsushi; Saito, Masayuki; Jin, Cheng-Hao; Mukasa, Chizu; Okabe, Taijiro; Nomura, Masatoshi; Goto, Kiminobu; Nawata, Hajime (Third Department of Internal Medicine, Faculty of Medicine, Kyushu University, Fukuoka, 812-8582, Japan). Endocrinology, 142(8), 3590-3597 (English) 2001. CODEN: ENDOAO. ISSN: 0013-7227. Publisher: Endocrine Society.

AB **Obesity** is associated with insulin resistance and some reproductive abnormalities. Circulating FFAs are often elevated in obese subjects and are also closely linked to insulin resistance. In this study, we demonstrated that saturated FFAs, such as palmitic acid and stearic acid, markedly suppressed the granulosa cell survival in a time- and dose-dependent manner. Polyunsatd. FFA, arachidonic acid, had no effect on the cell survival, even at supraphysiol. concns. The suppressive effect of saturated FFAs on cell survival was caused by apoptosis, as evidenced by DNA ladder formation and annexin V-EGFP/propidium iodide staining of the cells. The apoptotic effects of palmitic acid and stearic acid were unrelated to the increase of ceramide generation or nitric oxide production and were also completely blocked by Triacsin C, an inhibitor of acylCoA synthetase. In addition, acylCoA, palmitoylCoA, and stearylCoA markedly suppressed granulosa cell survival, whereas arachidonoylCoA had no such effect, and this finding was consistent with the effect of the resp. FFA form. Surprisingly, arachidonic acid instead showed a protective effect on palmitic acid- and stearic acid-induced cell apoptosis. A Western blot anal. showed the apoptosis of the granulosa cells induced by palmitic acid to be accompanied by the down-regulation of an apoptosis inhibitor, Bcl-2, and the up-regulation of an apoptosis effector, Bax. These results indicate that saturated FFAs induce apoptosis in human granulosa cells caused by the metabolism of the resp. acylCoA form, and the actual composition of circulating FFAs may thus play a critical role in the apoptotic events of human granulosa cells. These effects of FFAs on granulosa cell survival may be a possible mechanism for reproductive abnormalities, such as amenorrhea, which is frequently observed in obese women.

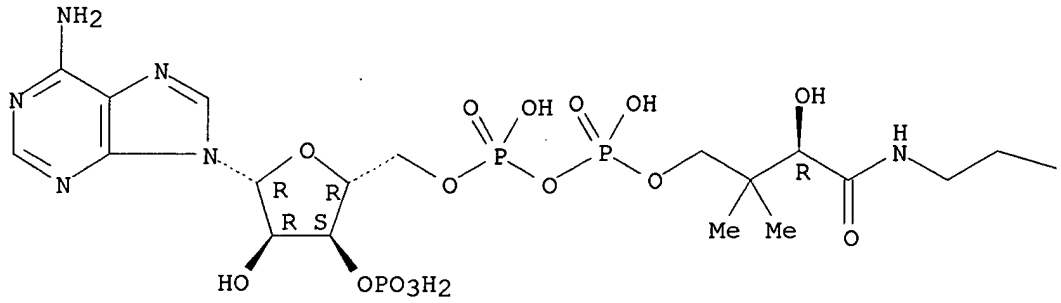
IT **362-66-3**, StearylCoA **1763-10-6**, PalmitoylCoA
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(palmitic acid and stearic acid induce apoptosis in human granulosa cells)

RN 362-66-3 CAPLUS

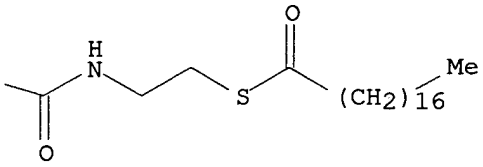
CN Coenzyme A, S-octadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



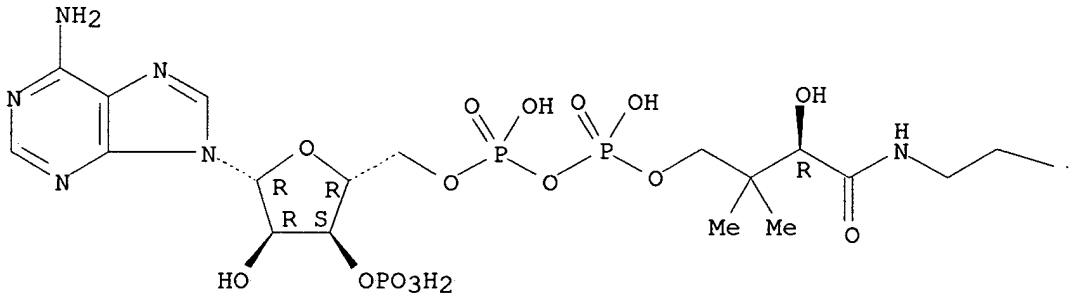
PAGE 1-B



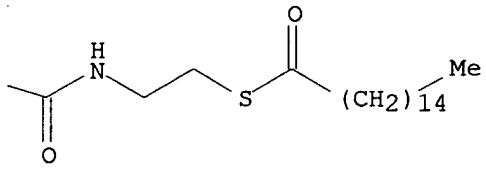
RN 1763-10-6 CAPLUS
CN Coenzyme A, S-hexadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



L9 ANSWER 32 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN
2001:507020 Document No. 135:341959 Effects of fatty acids on acyl-CoA thioesterase activity and expression of peroxisome proliferator activated receptors in human placental cells. Dutta-Roy, Asim K.; Crozet, Delphine; Taylor, Jonathon; Gordon, Margaret J. (Rowett Research Institute, Aberdeen, AB21 9SB, UK). γ-Linolenic Acid: Recent Advances in Biotechnology and Clinical Applications, [International Symposium on &gama;-Linolenic Acid], 2nd, San Diego, CA, United States, Apr. 25-28, 2000, Meeting Date 2000, 217-226. Editor(s): Huang, Yung-Sheng; Ziboh, Vincent A. AOCS Press: Champaign, Ill. (English) 2001. CODEN: 69BLYV.

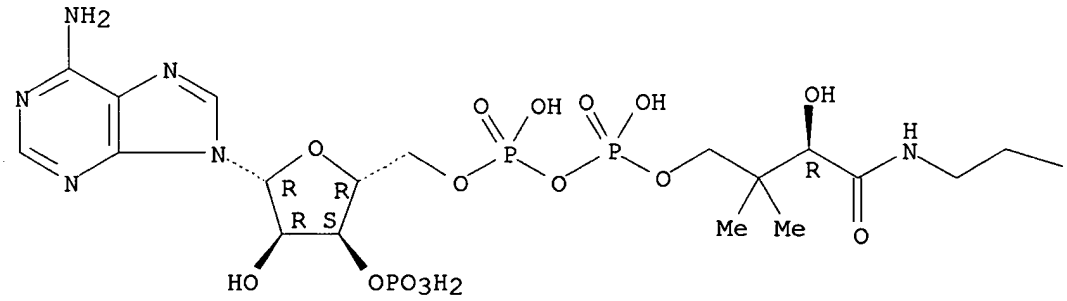
AB The human placental choriocarcinoma (BeWo) cells express acyl-CoA thioesterase activity and **PPAR**-γ. Preferred substrates for acyl-CoA thioesterase activity in these cells were γ-linolenoyl CoA, followed by arachidonoyl CoA, palmitoyl CoA, and linoleoyl CoA. Acyl-CoA thioesterase activity expressed by BeWo cells incubated with fatty acids (oleic acid (OA), eicosapentaenoic acid (EPA), docosahexaenoic acid (DHA), arachidonic acid (AA), conjugated linoleic acid (CLA), γ-linolenic acid (GLA)) bound to BSA solns. were measured using palmitoyl-CoA as substrate. The results showed that EPA, DHA and OA had no effect on acyl-CoA thioesterase activity compared with controls. But both CLA and GLA increased acyl-CoA thioesterase activity compared with controls. **PPAR**.gamma. expression was also affected by the fatty acids. Expression of **PPAR**.gamma. was higher in cells incubated with AA or GLA, compared with the control. Expts. were also carried out using the MTT assay to monitor fatty acid, prostaglandin, and ciglitazone-induced changes in BeWo cells. In these cells, PGJ2 induced apoptosis as determined by MTT assay, whereas fatty acids had minimal effect. The role of fatty acids in placental expression of **PPAR**.gamma. as well as fetal and placental growth and development was discussed.

IT **1763-10-6**, Palmitoyl CoA **6709-57-5**, Linoleoyl CoA **17046-56-9**, Arachidonoyl CoA **27843-61-4**, γ-Linolenoyl CoA
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(effects of fatty acids on acyl-CoA thioesterase activity and expression of peroxisome proliferator activated receptors in human placental cells)

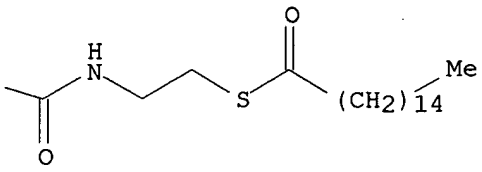
RN 1763-10-6 CAPLUS
CN Coenzyme A, S-hexadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



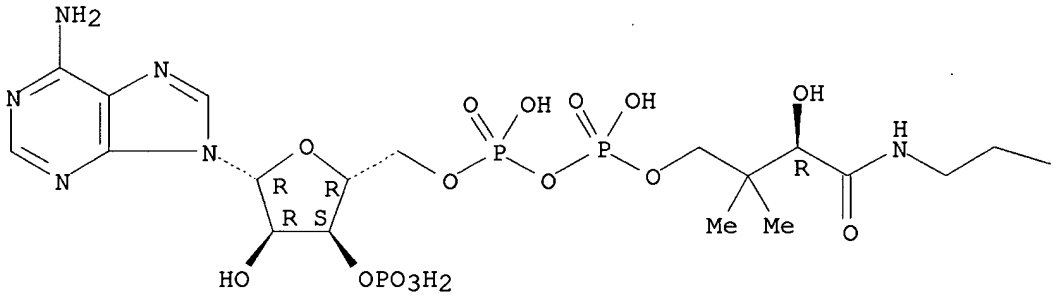
PAGE 1-B



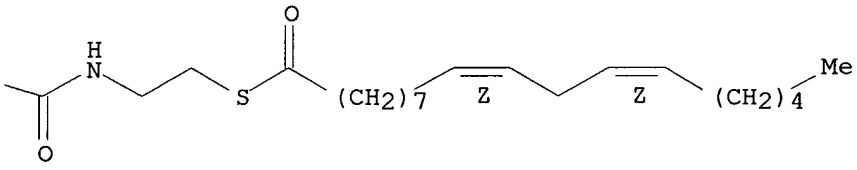
RN 6709-57-5 CAPLUS
CN Coenzyme A, S-(9Z,12Z)-9,12-octadecadienoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

PAGE 1-A



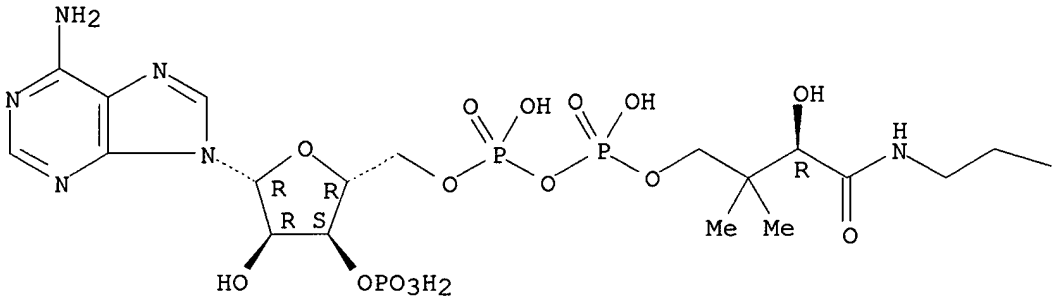
PAGE 1-B



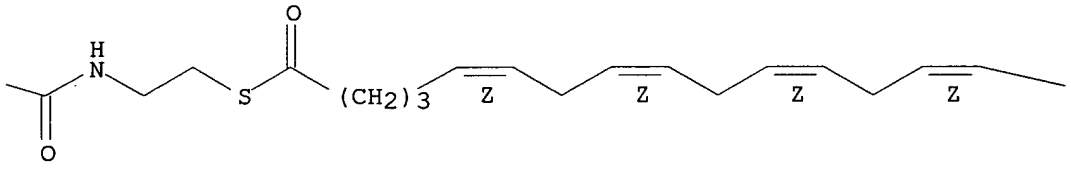
RN 17046-56-9 CAPLUS
CN Coenzyme A, S-(5Z,8Z,11Z,14Z)-5,8,11,14-eicosatetraenoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

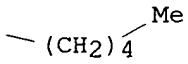
PAGE 1-A



PAGE 1-B



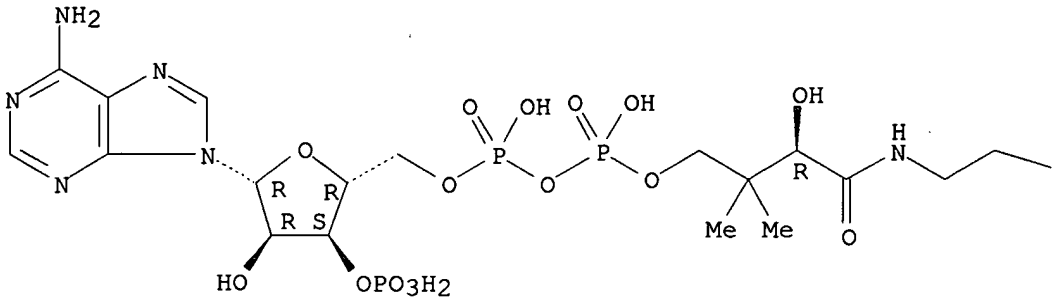
PAGE 1-C



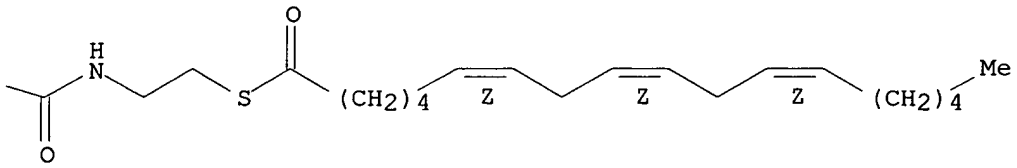
RN 27843-61-4 CAPLUS
CN Coenzyme A, S-(6Z,9Z,12Z)-6,9,12-octadecatrienoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

PAGE 1-A



PAGE 1-B



L9 ANSWER 33 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN
2001:455680 Document No. 135:162626 Acyl-CoA esters antagonize the effects of ligands on peroxisome proliferator-activated receptor α conformation, DNA binding, and interaction with Co-factors. Elholm, Morten; Dam, Inge; Jorgensen, Claus; Krogsdam, Anne-M.; Holst, Dorte; Kratchmarova, Irina; Gottlicher, Martin; Gustafsson, Jan-Ake; Berge, Rolf; Flatmark, Torgeir; Knudsen, Jens; Mandrup, Susanne; Kristiansen, Karsten (Department of Biochemistry and Molecular Biology and Center for Experimental BioInformatics, University of Southern Denmark, Odense, DK-5320, Den.). Journal of Biological Chemistry, 276(24), 21410-21416 (English) 2001. CODEN: JBCHA3. ISSN: 0021-9258. Publisher: American Society for Biochemistry and Molecular Biology.

AB The peroxisome proliferator-activated receptor α (**PPAR** α) is a ligand-activated transcription factor and a key regulator of lipid homeostasis. Numerous fatty acids and eicosanoids serve as ligands and activators for **PPAR**.alpha.. Here S-hexadecyl-CoA, a nonhydrolyzable palmitoyl-CoA analog, antagonizes the effects of agonists on **PPAR**.alpha. conformation and function in vitro. In electrophoretic mobility shift assays, S-hexadecyl-CoA prevented agonist-induced binding of the **PPAR**.alpha.-retinoid X receptor α heterodimer to the acyl-CoA oxidase peroxisome proliferator response element. **PPAR**.alpha. bound specifically to immobilized palmitoyl-CoA and Wy14643, but not BRL49653, abolished binding. S-Hexadecyl-CoA increased in a dose-dependent and reversible manner the sensitivity of **PPAR**.alpha. to chymotrypsin digestion, and the S-hexadecyl-CoA-induced sensitivity required a functional **PPAR** α ligand-binding pocket. S-Hexadecyl-CoA prevented ligand-induced interaction between the co-activator SRC-1 and **PPAR**.alpha. but increased recruitment of the nuclear receptor co-repressor NCoR. In

cells, the concentration of free acyl-CoA esters is kept in the low nanomolar range due to the buffering effect of high affinity acyl-CoA-binding proteins, especially the acyl-CoA-binding protein. By using **PPAR** α expressed in Sf21 cells for electrophoretic mobility shift assays, the authors demonstrate that S-hexadecyl-CoA was able to increase the mobility of the **PPAR**.alpha.-containing heterodimer even in the presence of a molar excess of acyl-CoA-binding protein, mimicking the conditions found in vivo.

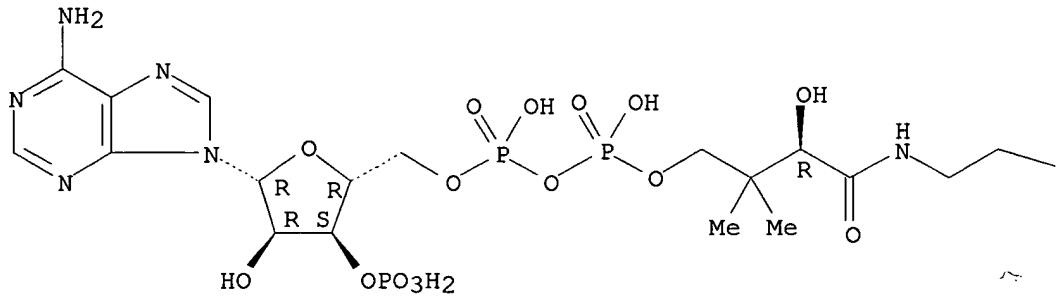
IT **1763-10-6**, palmitoyl-CoA
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(acyl-CoA esters antagonize effects of ligands on peroxisome proliferator-activated receptor α conformation and DNA binding and interaction with cofactors)

RN 1763-10-6 CAPLUS

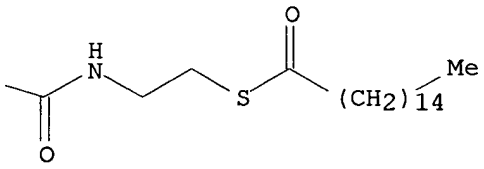
CN Coenzyme A, S-hexadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



L9 ANSWER 34 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN

2001:259942 Document No. 135:86962 Suppression of hepatocyte nuclear factor-4α by acyl-CoA thioesters of hypolipidemic peroxisome proliferators. Hertz, R.; Sheena, V.; Kalderon, B.; Berman, I.; Bar-Tana, J. (Department of Human Nutrition and Metabolism, Hebrew University Medical School, Jerusalem, 91120, Israel). Biochemical Pharmacology, 61(9), 1057-1062 (English) 2001. CODEN: BCPCA6. ISSN: 0006-2952. Publisher: Elsevier Science Inc..

AB Hepatocyte nuclear factor-4α (HNF-4α) modulates the expression of liver-specific genes that control the production (e.g. apolipoprotein [apo] A-I and apo B) and clearance (e.g. apo C-III) of plasma lipoproteins. We

reported that the CoA thioesters of amphipathic carboxylic hypolipidemic drugs (e.g. clofibric acid analogs currently used for treating hyperlipidemia in humans and substituted long-chain dicarboxylic acids) were formed in vivo, bound to HNF-4α, inhibited its transcriptional activity, and suppressed the expression of HNF-4α-responsive genes. Hypolipidemic **PPAR**.alpha. (peroxisome proliferator-activated receptor alpha) activators that were not endogenously thioesterified into their resp. acyl-CoAs were shown to be effective in rats but not in humans, implying that the hypolipidemic activity transduced by **PPAR**.alpha. in rats was **PPAR**.alpha.-independent in humans. The suppressed acyl-CoA synthase of **PPAR**.alpha. knockout mice left unresolved the contribution made by the acyl-CoA/HNF-4α pathway to the hypolipidemic effect of **PPAR** α agonists in rodents. Hence, suppression of HNF-4α activity by the CoA thioesters of hypolipidemic "peroxisome proliferators" may account for their hypolipidemic activity independently of **PPAR** α activation by their resp. free carboxylates. The hypolipidemic activity of peroxisome proliferators is mediated in rats and humans by the **PPAR**.alpha. and HNF-4α pathways, resp.

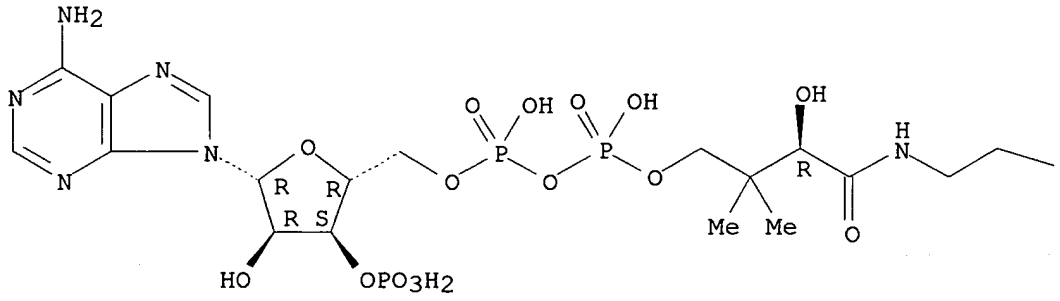
IT **349482-27-5 349482-28-6 349482-29-7 349482-30-0D**, homologs **349482-31-1**
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(hypolipidemic activity of peroxisome proliferators is mediated in rats and humans by the **PPAR**.alpha. and HNF-4α pathways, resp.)

RN 349482-27-5 CAPLUS

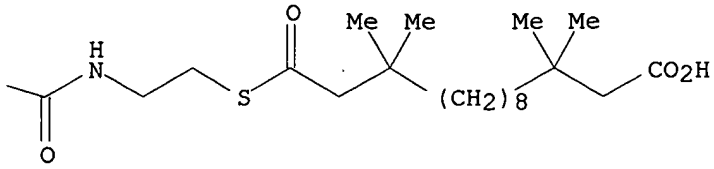
CN Coenzyme A, S-(hydrogen 3,3,12,12-tetramethyltetradecanedioate) (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



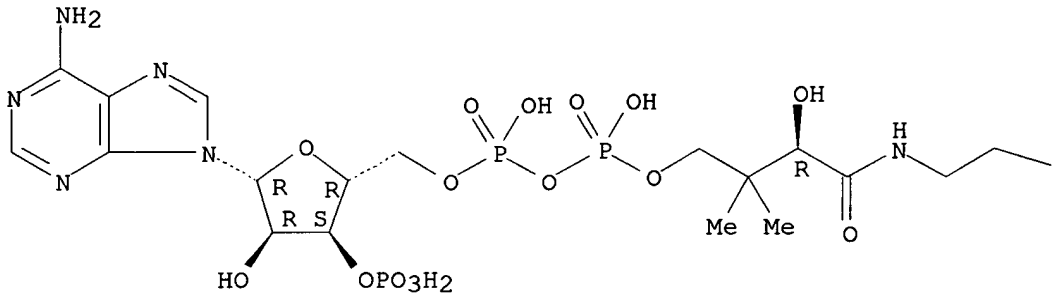
PAGE 1-B



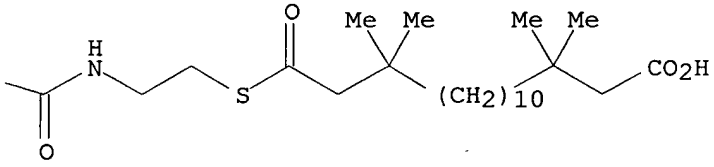
RN 349482-28-6 CAPLUS
CN Coenzyme A, S-(hydrogen 3,3,14,14-tetramethylhexadecanedioate) (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



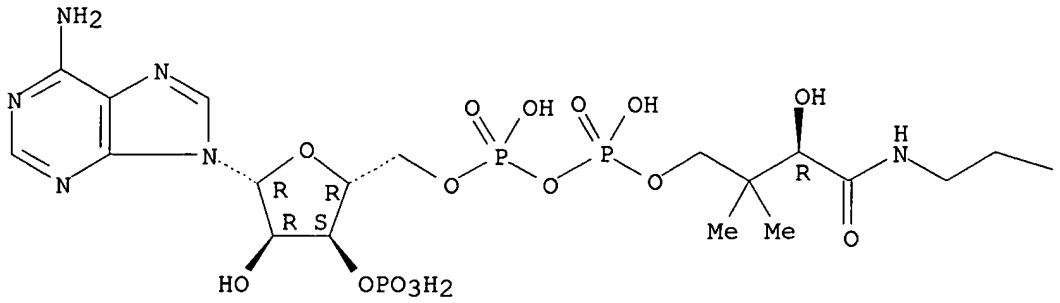
PAGE 1-B



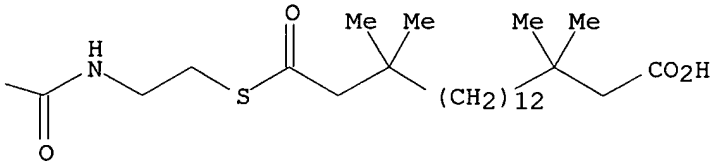
RN 349482-29-7 CAPLUS
CN Coenzyme A, S-(hydrogen 3,3,16,16-tetramethyloctadecanedioate) (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



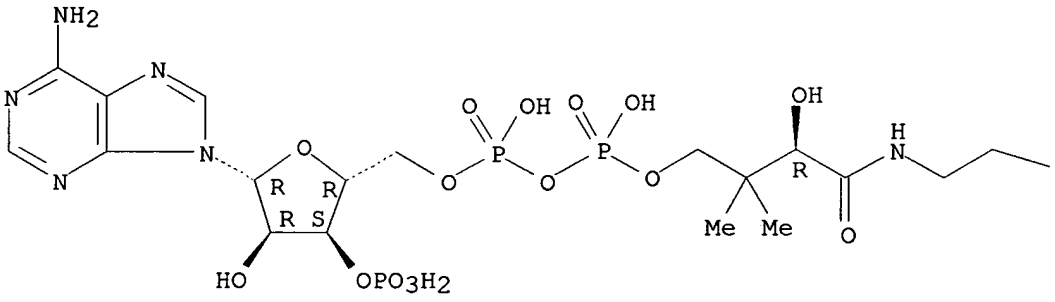
PAGE 1-B



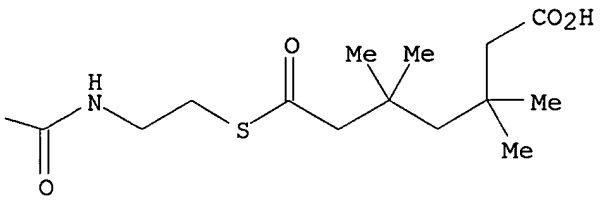
RN 349482-30-0 CAPLUS
CN Coenzyme A, S-(hydrogen 3,3,5,5-tetramethylheptanedioate) (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



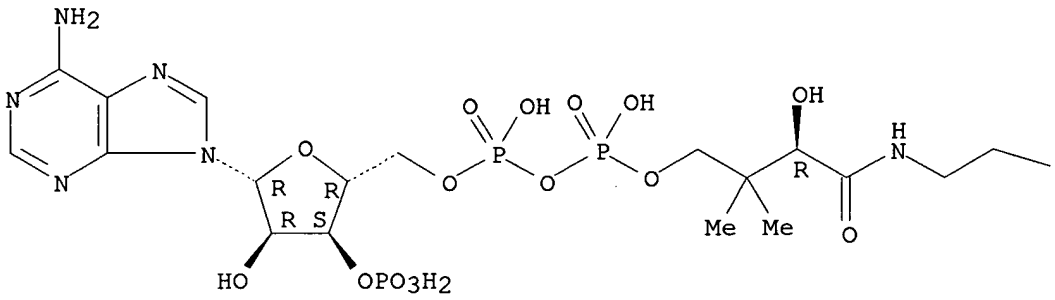
PAGE 1-B



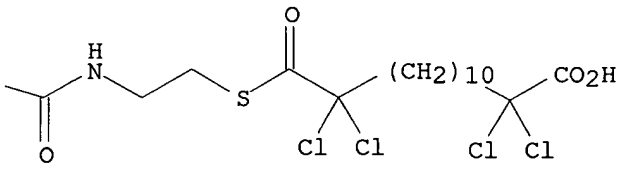
RN 349482-31-1 CAPLUS
CN Coenzyme A, S-(hydrogen 2,2,13,13-tetrachlorotetradecanedioate) (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



L9 ANSWER 35 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN
2001:81138 Document No. 134:248612 Fatty-acyl-CoA thioesters inhibit recruitment of steroid receptor co-activator 1 to α and γ isoforms of peroxisome-proliferator-activated receptors by competing with agonists. Murakami, Koji; Ide, Tomohiro; Nakazawa, Tomoko; Okazaki, Takashi; Mochizuki, Toshiro; Kadowaki, Takashi (Central Research Laboratories, Kyorin Pharmaceutical Co., Ltd., Tochigi, 329-0114, Japan). Biochemical Journal, 353(2), 231-238 (English) 2001. CODEN: BIJOAK. ISSN: 0264-6021. Publisher: Portland Press Ltd..
AB Peroxisome-proliferator-activated receptors (PPARs) α and γ are ligand-dependent transcription factors that are key regulators of

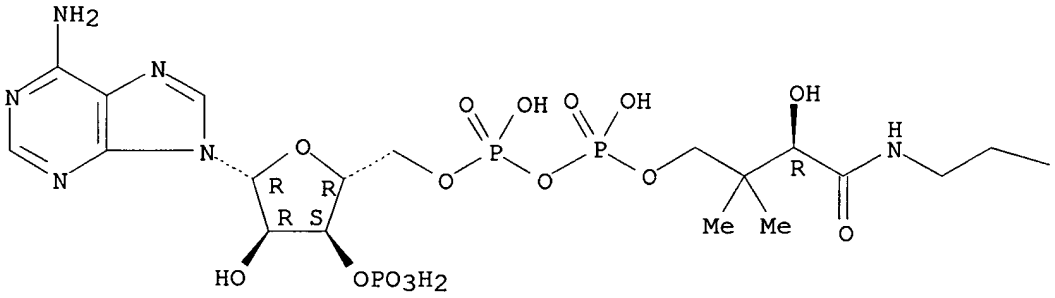
lipid and carbohydrate homeostasis. Fatty acids bind to the ligand-binding domains (LBDs) of **PPAR**.alpha. and **PPAR** γ and activate these receptors. To clarify whether fatty-acyl-CoAs interact directly with the LBDs of **PPAR**.alpha. and **PPAR** γ, we performed a competition binding assay with radiolabeled KRP-297, a known dual agonist for these receptors. We show here that fatty-acyl-CoAs bind directly to **PPAR**.alpha. and **PPAR** γ. Interestingly, fatty-acyl-CoAs, unlike fatty acids, failed to recruit steroid receptor co-activator 1 (SRC-1), on the basis of conformational changes in the LBDs of **PPAR**.alpha. and **PPAR**.gamma.. Moreover, fatty-acyl-CoAs also markedly inhibited agonist-induced recruitment of SRC-1. These findings demonstrate that fatty-acyl-CoAs have a novel function in the signaling pathways of **PPAR**.alpha. and **PPAR**.gamma..

IT **362-66-3**, Stearoyl-CoA **1716-06-9**, Oleoyl-CoA
1763-10-6, Palmitoyl-CoA **3130-72-1**, Myristoyl-CoA
6709-57-5, Linoleoyl-CoA **17046-56-9**, Arachidonoyl-CoA
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(fatty acyl-CoA thioesters inhibit agonist-induced recruitment of steroid receptor co-activator 1 to peroxisome-proliferator-activated receptors α and γ)

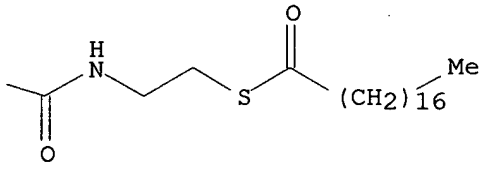
RN 362-66-3 CAPLUS
CN Coenzyme A, S-octadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B

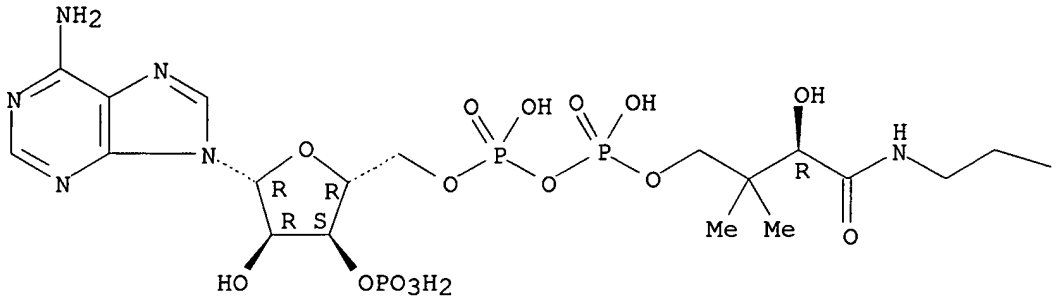


RN 1716-06-9 CAPLUS
CN Coenzyme A, S-(9Z)-9-octadecenoate (9CI) (CA INDEX NAME)

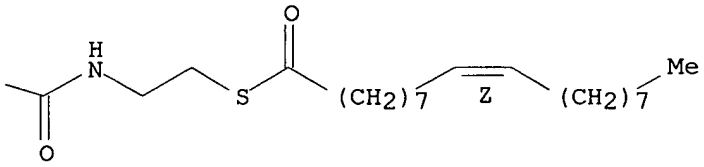
Absolute stereochemistry.

Double bond geometry as shown.

PAGE 1-A



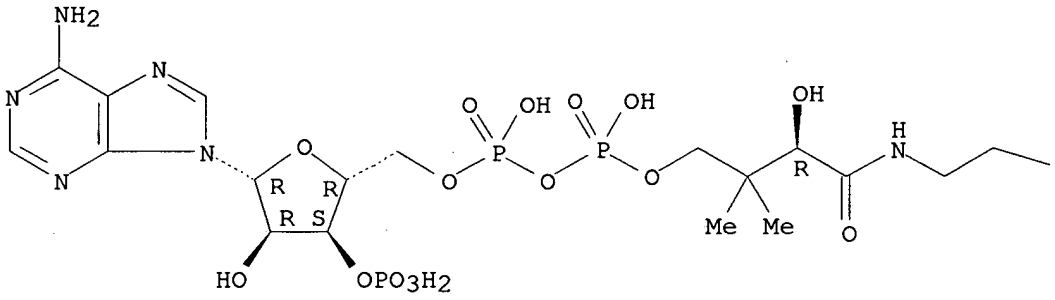
PAGE 1-B



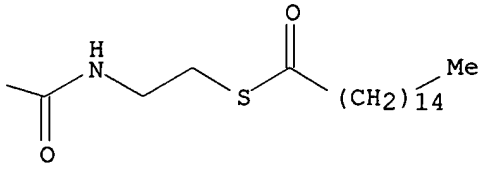
RN 1763-10-6 CAPLUS
CN Coenzyme A, S-hexadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



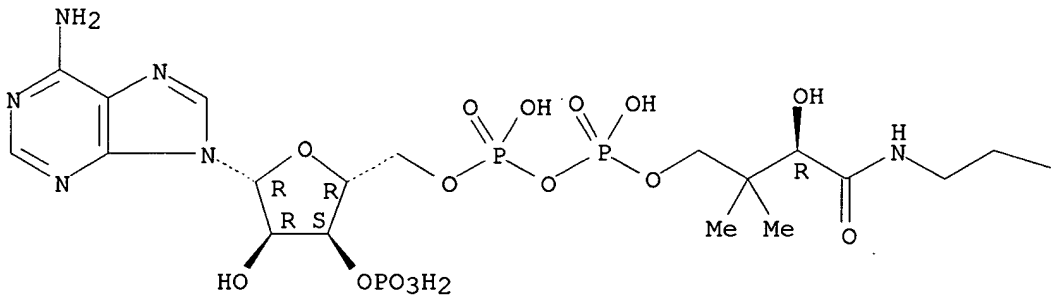
PAGE 1-B



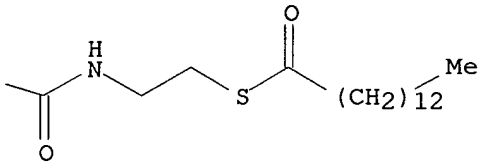
RN 3130-72-1 CAPLUS
CN Coenzyme A, S-tetradecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



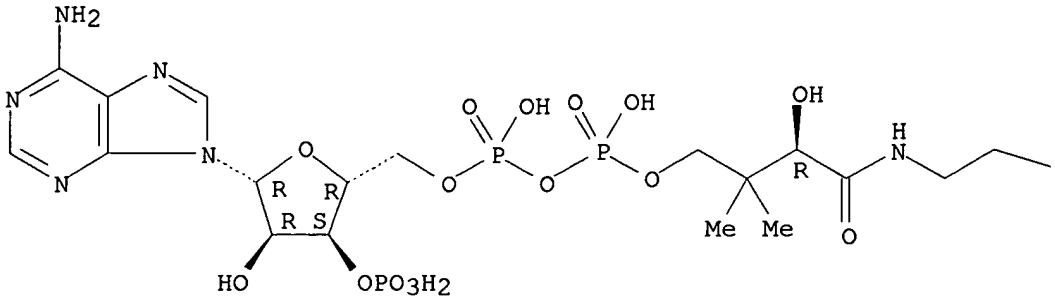
PAGE 1-B



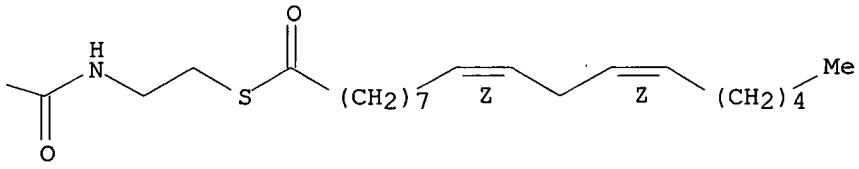
RN 6709-57-5 CAPLUS
CN Coenzyme A, S-(9Z,12Z)-9,12-octadecadienoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

PAGE 1-A



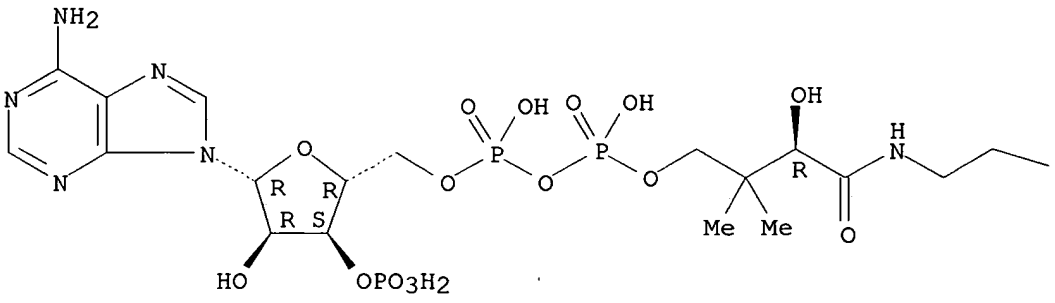
PAGE 1-B



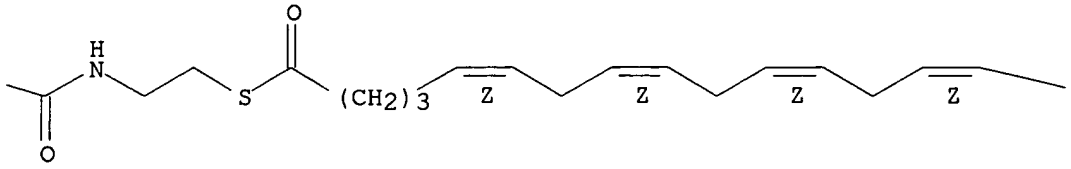
RN 17046-56-9 CAPLUS
CN Coenzyme A, S-(5Z,8Z,11Z,14Z)-5,8,11,14-eicosatetraenoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

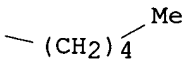
PAGE 1-A



PAGE 1-B



PAGE 1-C



L9 ANSWER 36 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN

2002:775210 Document No. 138:23163 Effect of weight loss on insulin sensitivity and intramuscular long-chain fatty acyl-CoAs in morbidly obese subjects. Houmard, Joseph A.; Tanner, Charles J.; Yu, Chunli; Cunningham, Paul G.; Pories, Walter J.; MacDonald, Kenneth G.; Shulman, Gerald I. (Departments of Exercise and Sport Science, Surgery, East Carolina University, Greenville, NC, 27858, USA). Diabetes, 51(10), 2959-2963 (English) 2002. CODEN: DIAEAZ. ISSN: 0012-1797. Publisher: American Diabetes Association.

AB Increases in intramyocellular long-chain fatty acyl-CoAs (LCACoA) have been implicated in the pathogenesis of insulin resistance in skeletal muscle. To test this hypothesis, we measured muscle (vastus lateralis) LCACoA content and insulin action in morbidly obese patients (n = 11) before and after weight loss (gastric bypass surgery). The intervention produced significant weight loss (142.3±6.8 vs. 79.6±4.1 kg for before vs. after surgery, resp.). Fasting insulin decreased by .apprx.84% (23.3±3.8 vs. 3.8±0.5 mU/mL), and insulin sensitivity, as determined by minimal model, increased by .apprx.360% (1.2±0.3 vs. 4.1±0.5 min-1 · [μU/kg-1]) indicating enhanced insulin action. Muscle palmityl CoA (16:0; 0.54±0.08 vs. 0.35±0.04 nmol/g wet weight) concentration decreased by .apprx.35% ($P < 0.05$) with weight loss, whereas stearate CoA (18:0; -17%; 0.65±0.05 vs. 0.54±0.03 nmol/g wet weight) and linoleate CoA (18:2; -30%; 2.47±0.27 vs. 1.66±0.19 nmol/g wet weight) were also reduced ($P < 0.05$). There were no statistically significant declines in muscle palmitoleate CoA (16:1), oleate CoA (18:1), or total LCACoA content. These data suggest that a reduction in i.m. LCACoA content may be responsible, at least in part, for the enhanced insulin action observed with weight loss in obese individuals.

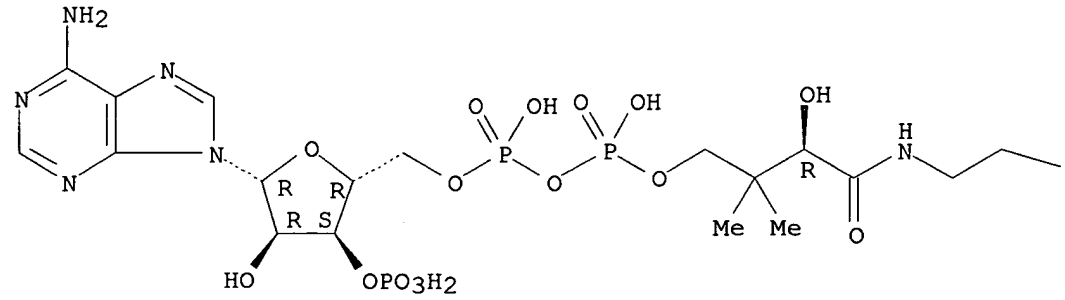
IT **362-66-3**, Stearoyl coenzyme A **1763-10-6**, Palmityl coenzyme A **6709-57-5**, Linoleoyl coenzyme A
RL: BSU (Biological study, unclassified); BIOL (Biological study)

(effect of weight loss on insulin sensitivity and i.m. long-chain fatty acyl-CoAs in morbidly obese subjects)

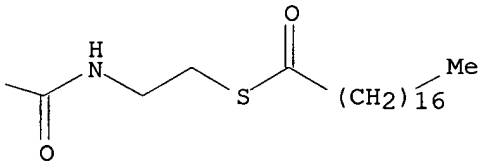
RN 362-66-3 CAPLUS
CN Coenzyme A, S-octadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



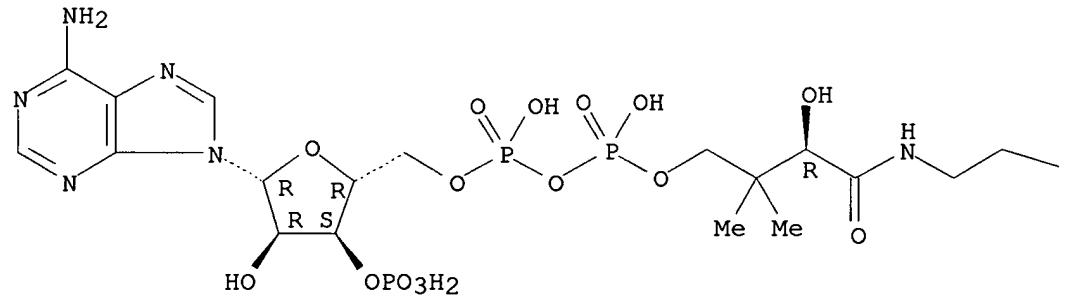
PAGE 1-B



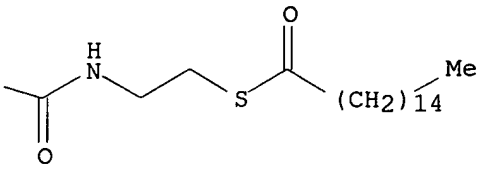
RN 1763-10-6 CAPLUS
CN Coenzyme A, S-hexadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



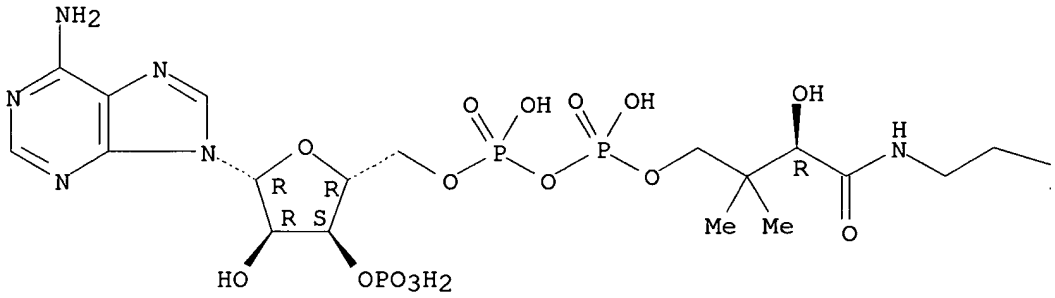
PAGE 1-B



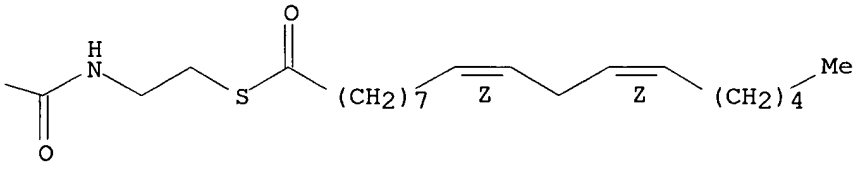
RN 6709-57-5 CAPLUS
CN Coenzyme A, S-(9Z,12Z)-9,12-octadecadienoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

PAGE 1-A



PAGE 1-B



L9 ANSWER 37 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN
2002:744054 Document No. 138:22939 Oleate and linoleate enhance the growth-promoting effects of IGF-I through a Phospholipase D-dependent pathway in arterial smooth muscle cells. Askari, Bardia; Carroll, Mairead A.; Capparelli, Maria; Kramer, Farah; Gerrity, Ross G.; Bornfeldt, Karin E. (Department of Pathology, University of Washington School of Medicine, Seattle, WA, 98195, USA). Journal of Biological Chemistry, 277(39), 36338-36344 (English) 2002. CODEN: JBCHA3. ISSN: 0021-9258. Publisher: American Society for Biochemistry and Molecular Biology.
AB **Diabetes** causes accelerated atherosclerosis and subsequent cardiovascular disease through mechanisms that are poorly understood. We

have previously shown, using a porcine model of **diabetes** -accelerated atherosclerosis, that **diabetes** leads to an increased accumulation and proliferation of arterial smooth muscle cells in atherosclerotic lesions and that this is associated with elevated levels of plasma triglycerides. We therefore used the same model to investigate the mechanism whereby **diabetes** may stimulate smooth muscle cell proliferation. We show that lesions from diabetic pigs fed a cholesterol-rich diet contain abundant insulin-like growth factor-I (IGF-I), in contrast to lesions from non-diabetic pigs. Furthermore, two fatty acids common in triglycerides, oleate and linoleate, enhance the growth-promoting effects of IGF-I in smooth muscle cells isolated from these animals. These fatty acids accumulate predominantly in the membrane phospholipid pool; oleate accumulates preferentially in phosphatidylcholine and phosphatidylethanolamine, whereas linoleate is found mainly in phosphatidylethanolamine. The growth-promoting effects of oleate and linoleate depend on phospholipid hydrolysis by phospholipase D and subsequent generation of diacylglycerol. Thus, concurrent increases in levels of IGF-I and triglyceride-derived oleate and linoleate in lesions may contribute to accumulation and proliferation of smooth muscle cells and lesion progression in **diabetes**-accelerated atherosclerosis.

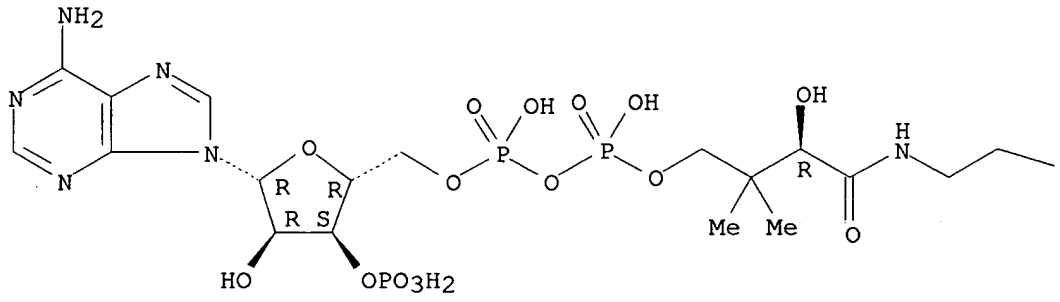
IT **1716-06-9D**, Oleoyl-CoA, ester **6709-57-5D**, Linoleyl-CoA, ester
RL: ADV (Adverse effect, including toxicity); BSU (Biological study, unclassified); BIOL (Biological study)
(oleate and linoleate-induced growth-promoting effects of IGF-I via phospholipase D pathway in arterial smooth muscle in **diabetes** -accelerated atherosclerosis)

RN 1716-06-9 CAPLUS

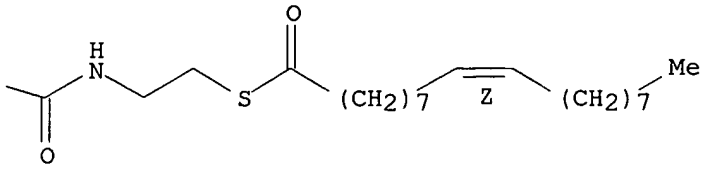
CN Coenzyme A, S-(9Z)-9-octadecenoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

PAGE 1-A



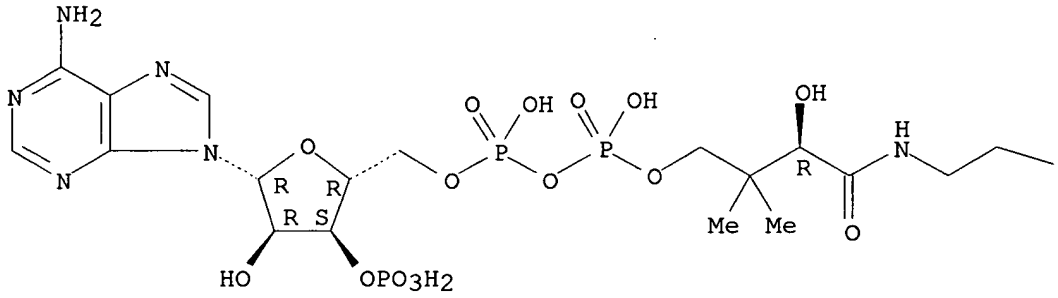
PAGE 1-B



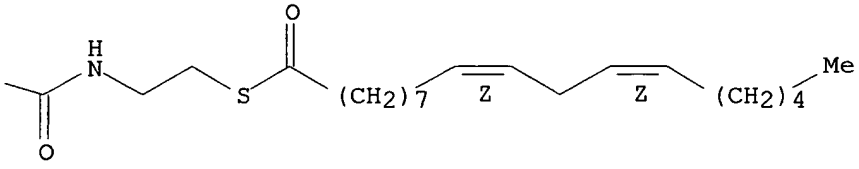
RN 6709-57-5 CAPLUS
CN Coenzyme A, S-(9Z,12Z)-9,12-octadecadienoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

PAGE 1-A



PAGE 1-B



L9 ANSWER 38 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN
2002:133198 Document No. 136:294126 Skeletal muscle oxidative capacity in rats fed high-fat diet. Iossa, S.; Mollica, M. P.; Lionetti, L.; Crescenzo, R.; Botta, M.; Liverini, G. (Department of General and Environmental Physiology, University of Naples "Federico II", Naples, Italy). International Journal of Obesity, 26(1), 65-72 (English) 2002. CODEN: IJOBDP. ISSN: 0307-0565. Publisher: Nature Publishing Group.
AB Objective: To investigate whether young rats respond to high-fat feeding through changes in energy efficiency and fuel partitioning at the level of skeletal muscle, to avoid **obesity** development. In addition, to establish whether the two mitochondrial subpopulations, which exist in

skeletal muscle, ie subsarcolemmal and intermyofibrillar, are differently affected by high-fat feeding. Design: Weaning rats were fed a low-fat or a high-fat diet for 15 days. Measurements: Energy balance and lipid partitioning in the whole animal. State 3 and state 4 oxygen consumption rates in whole skeletal muscle homogenate. State 3 and state 4 oxygen consumption rates, membrane potential and uncoupling effect of palmitate in subsarcolemmal and intermyofibrillar mitochondria from skeletal muscle. Results: Rats fed a high-fat diet showed an increased whole body lipid utilization. Skeletal muscle NAD-linked and lipid oxidative capacity significantly increased at the whole-tissue level, due to an increase in lipid oxidative capacity in subsarcolemmal and intermyofibrillar mitochondria and in NAD-linked activity only in intermyofibrillar ones. In addition, rats fed a high-fat diet showed an increase in the uncoupling effect of palmitate in both the mitochondrial populations. Conclusions: In young rats fed a high-fat diet, skeletal muscle contributes to enhanced whole body lipid oxidation through an increased mitochondrial capacity to use lipids as metabolic fuels, associated with a decrease in energy coupling.

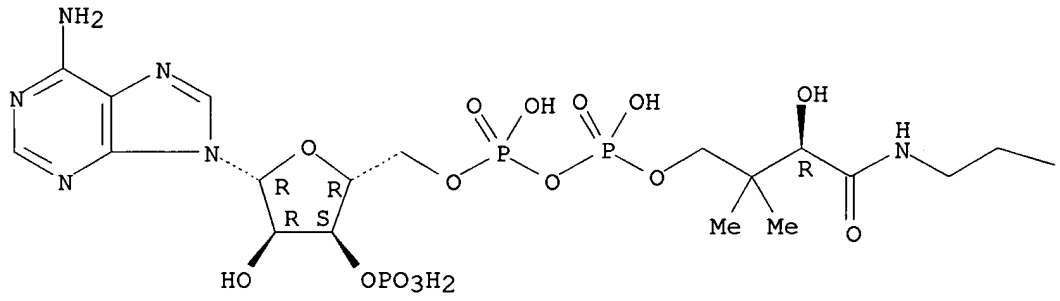
IT **1763-10-6**, Palmitoyl CoA
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(skeletal muscle oxidative capacity in rats fed high-fat diet)

RN 1763-10-6 CAPLUS

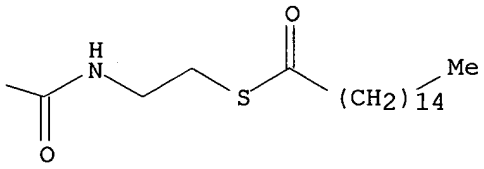
CN Coenzyme A, S-hexadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



L9 ANSWER 39 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN

2002:102997 Document No. 136:292797 Acyl-CoA binding protein expression is fiber type-specific and elevated in muscles from the obese insulin-resistant Zucker rat. Franch, Jesper; Knudsen, Jens; Ellis, Bronwyn A.; Pedersen, Preben K.; Cooney, Gregory J.; Jensen, Jorgen

(Institute of Sports Science and Clinical Biomechanics, University of Southern Denmark, Odense, Den.). Diabetes, 51(2), 449-454 (English) 2002. CODEN: DIAEAZ. ISSN: 0012-1797. Publisher: American Diabetes Association.

AB Accumulation of acyl-CoA is hypothesized to be involved in development of insulin resistance. Acyl-CoA binds to acyl-CoA binding protein (ACBP) with high affinity, and therefore knowledge about ACBP concentration is important for interpreting acyl-CoA data. In the present study, we used a sandwich ELISA to quantify ACBP concentration in different muscle fiber types. Furthermore, ACBP concentration was compared in muscles from lean and obese Zucker rats. Expression of ACBP was highest in the slow-twitch oxidative soleus muscle and lowest in the fast-twitch glycolytic white gastrocnemius (0.46±0.02 and 0.16±0.005 μg/mg protein, resp.). Expression of ACBP was soleus > red gastrocnemius > extensor digitorum longus > white gastrocnemius. Similar fiber type differences were found for carnitine palmitoyl transferase (CPT)-1, and a correlation was observed between ACBP and CPT-1. Muscles from obese Zucker rats had twice the triglyceride content, had approx. twice the long-chain acyl CoA content, and were severely insulin resistant. ACBP concentration was .apprx.30% higher in all muscles from obese rats. Activities of CPT-1 and 3-hydroxy-acyl-CoA dehydrogenase were increased in muscles from obese rats, whereas citrate synthase activity was similar. In conclusion, ACBP expression is fiber type-specific with the highest concentration in oxidative muscles and the lowest in glycolytic muscles. The 90% increase in the concentration of acyl-CoA in obese Zucker muscle compared with only a 30% increase in the concentration of ACBP supports the hypothesis that an increased concentration of free acyl-CoA is involved in the development of insulin resistance.

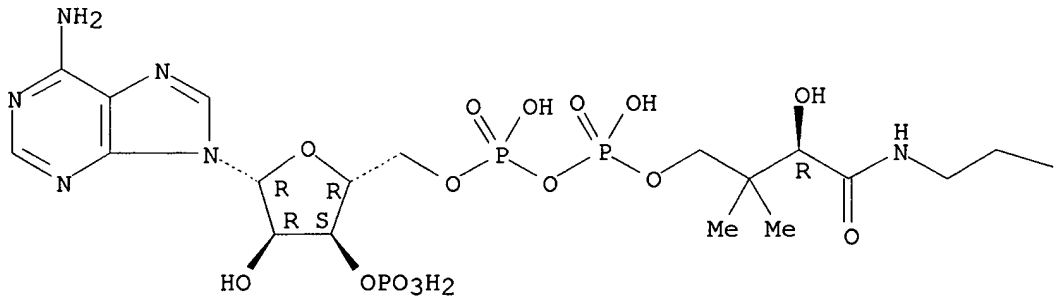
IT **1716-06-9**, Oleoyl coenzyme A **1763-10-6**, Palmitoyl coenzyme A **6709-57-5**, Linoleoyl coenzyme A
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(acyl-CoA binding protein expression is fiber type-specific and elevated in muscles from obese insulin-resistant Zucker rat)

RN 1716-06-9 CAPLUS

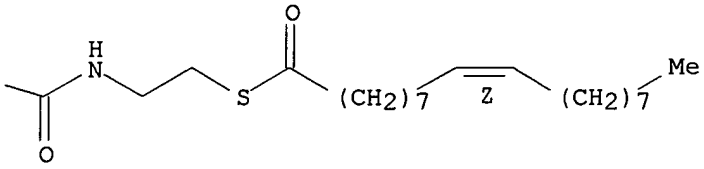
CN Coenzyme A, S-(9Z)-9-octadecenoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

PAGE 1-A



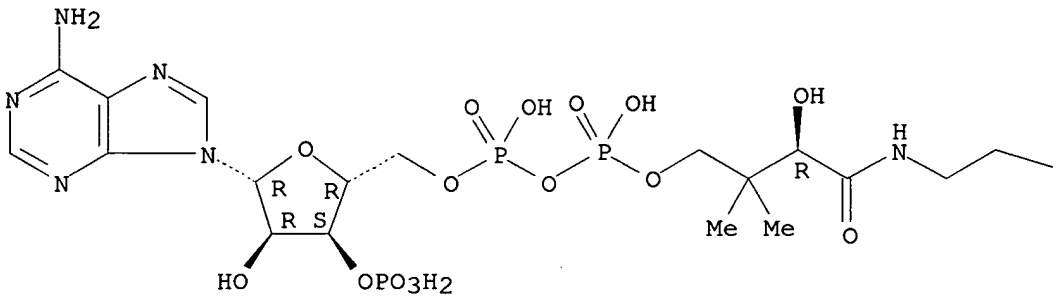
PAGE 1-B



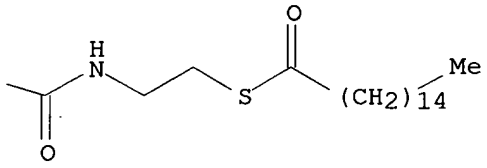
RN 1763-10-6 CAPLUS
CN Coenzyme A, S-hexadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



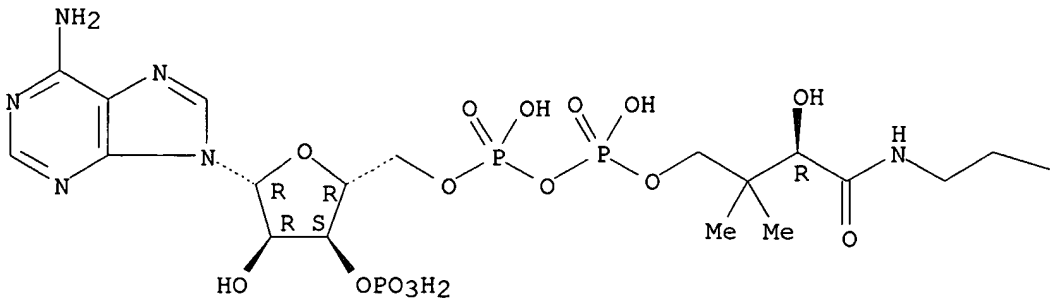
PAGE 1-B



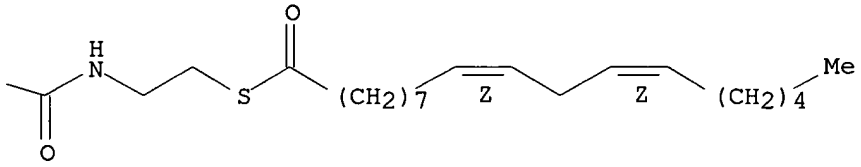
RN 6709-57-5 CAPLUS
CN Coenzyme A, S-(9Z,12Z)-9,12-octadecadienoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

PAGE 1-A



PAGE 1-B



L9 ANSWER 40 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN

2002:79757 Document No. 137:17046 Characterization of an acyl-CoA thioesterase that functions as a major regulator of peroxisomal lipid metabolism. Hunt, Mary C.; Solaas, Karianne; Kase, B. Frode; Alexson, Stefan E. H. (Department of Medical Laboratory Sciences and Technology, Division of Clinical Chemistry, Karolinska Institutet, Huddinge University Hospital, Stockholm, SE-141 86, Swed.). Journal of Biological Chemistry, 277(2), 1128-1138 (English) 2002. CODEN: JBCHA3. ISSN: 0021-9258. Publisher: American Society for Biochemistry and Molecular Biology.

AB Peroxisomes function in β-oxidation of very long and long-chain fatty acids, dicarboxylic fatty acids, bile acid intermediates, prostaglandins, leukotrienes, thromboxanes, pristanic acid, and xenobiotic carboxylic acids. These lipids are mainly chain-shortened for excretion as the carboxylic acids or transported to mitochondria for further metabolism Several of these carboxylic acids are slowly oxidized and may therefore sequester CoA (CoASH). To prevent CoASH sequestration and to facilitate excretion of chain-shortened carboxylic acids, acyl-CoA thioesterases, which catalyze the hydrolysis of acyl-CoAs to the free acid and CoASH, may play important roles. Here we have cloned and characterized a peroxisomal acyl-CoA thioesterase from mouse, named PTE-2 (peroxisomal acyl-CoA thioesterase 2). PTE-2 is ubiquitously expressed and induced at mRNA level by treatment with the peroxisome proliferator WY-14,643 and fasting. Induction seen by these treatments was dependent on the peroxisome proliferator-activated receptor α. Recombinant PTE-2 showed a broad chain length specificity with acyl-CoAs from short- and medium-, to long-chain acyl-CoAs, and other substrates including trihydroxycoprostanoyl-CoA, hydroxymethylglutaryl-CoA, and branched chain acyl-CoAs, all of which are present in peroxisomes. Highest activities were found with the CoA esters of primary bile acids choloyl-CoA and

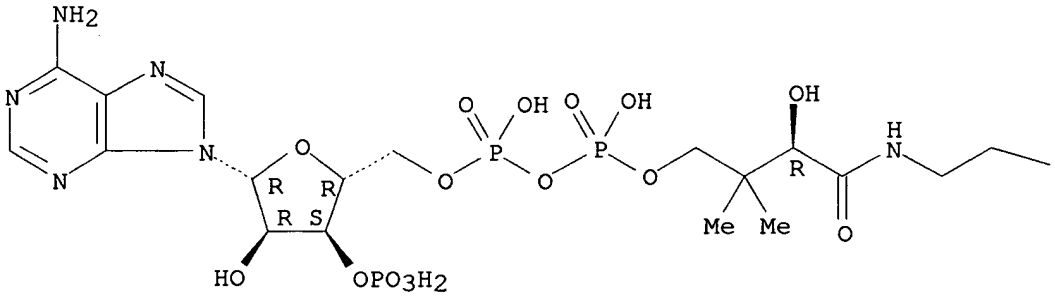
chenodeoxycholoyl-CoA as substrates. PTE-2 activity is inhibited by free CoASH, suggesting that intraperoxisomal free CoASH levels regulate the activity of this enzyme. The acyl-CoA specificity of recombinant PTE-2 closely resembles that of purified mouse liver peroxisomes, suggesting that PTE-2 is the major acyl-CoA thioesterase in peroxisomes. Addition of recombinant PTE-2 to incubations containing isolated mouse liver peroxisomes strongly inhibited bile acid-CoA:amino acid N-acyltransferase activity, suggesting that this thioesterase can interfere with CoASH-dependent pathways. We propose that PTE-2 functions as a key regulator of peroxisomal lipid metabolism

IT **362-66-3,** Stearoyl-CoA **1264-52-4,** Octanoyl-CoA **1264-57-9,** Decanoyl-CoA **1716-06-9,** Oleoyl-CoA **1763-10-6,** Palmitoyl-CoA **3130-72-1,** Myristoyl-CoA **5060-32-2,** Hexanoyl-CoA **6244-92-4,** Lauroyl-CoA **6709-57-5,** Linoleoyl-CoA **10018-95-8,** 2-trans-Decenoyl-CoA **15895-27-9,** Arachidoyl-CoA **17046-56-9,** Arachidonoyl-CoA **18198-76-0,** Palmitoleoyl-CoA **35106-50-4 57458-60-3 87935-97-5,** Myristoleoyl-CoA **204120-61-6 409307-21-7 434329-16-5**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(characterization of acyl-CoA thioesterase PTE-2 that functions as major regulator of peroxisomal lipid metabolism)

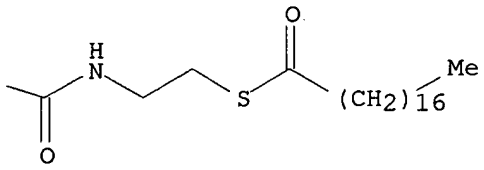
RN 362-66-3 CAPLUS
CN Coenzyme A, S-octadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



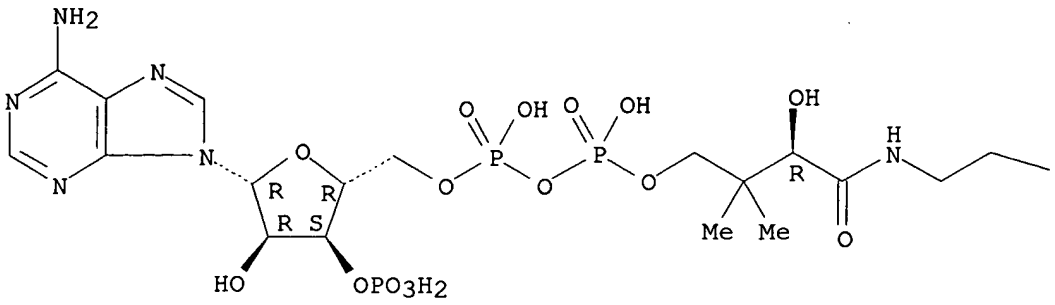
PAGE 1-B



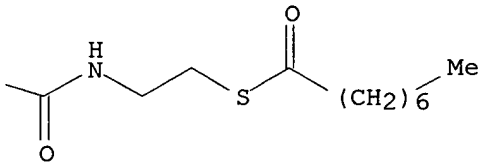
RN 1264-52-4 CAPLUS
CN Coenzyme A, S-octanoate (6CI, 7CI, 8CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



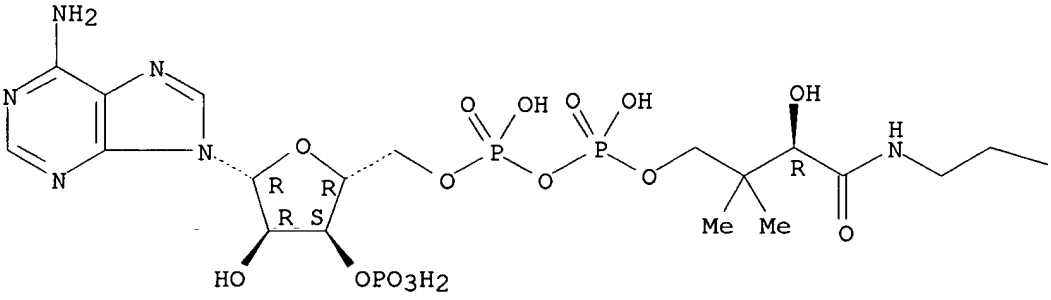
PAGE 1-B



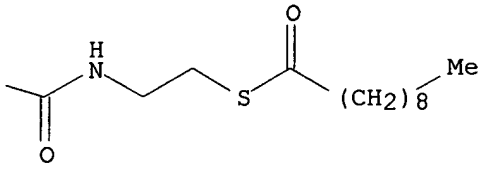
RN 1264-57-9 CAPLUS
CN Coenzyme A, S-decanoate (7CI, 8CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



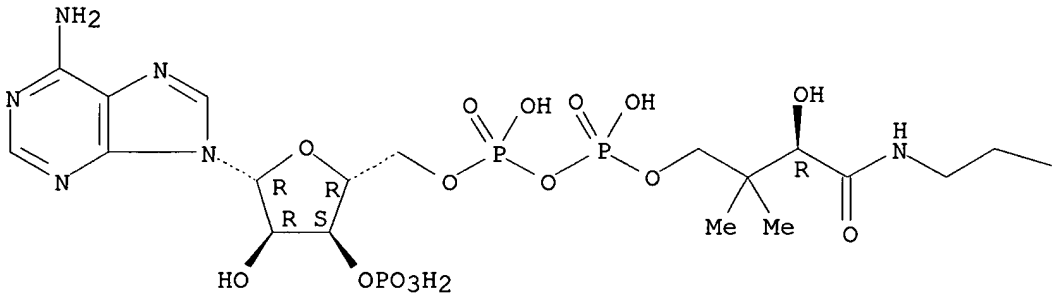
PAGE 1-B



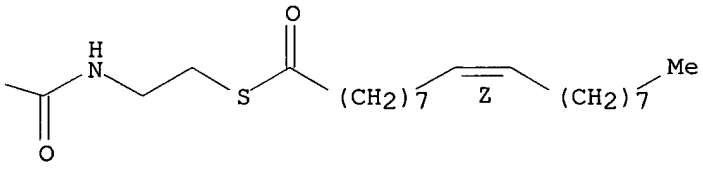
RN 1716-06-9 CAPLUS
CN Coenzyme A, S-(9Z)-9-octadecenoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

PAGE 1-A



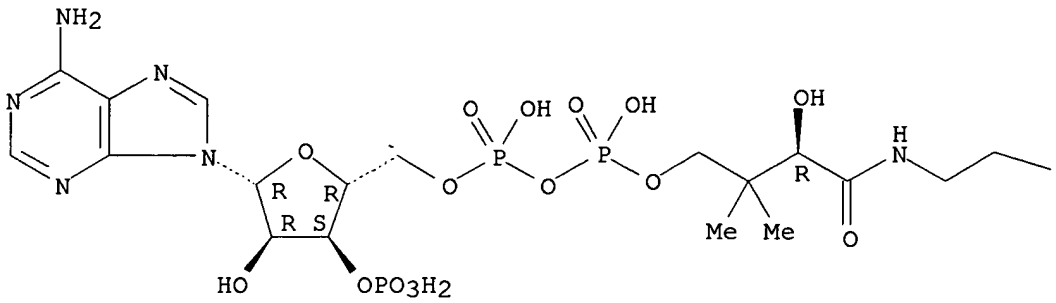
PAGE 1-B



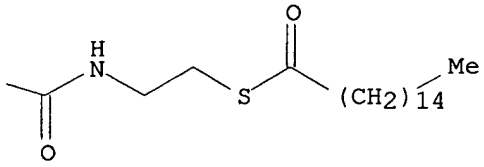
RN 1763-10-6 CAPLUS
CN Coenzyme A, S-hexadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



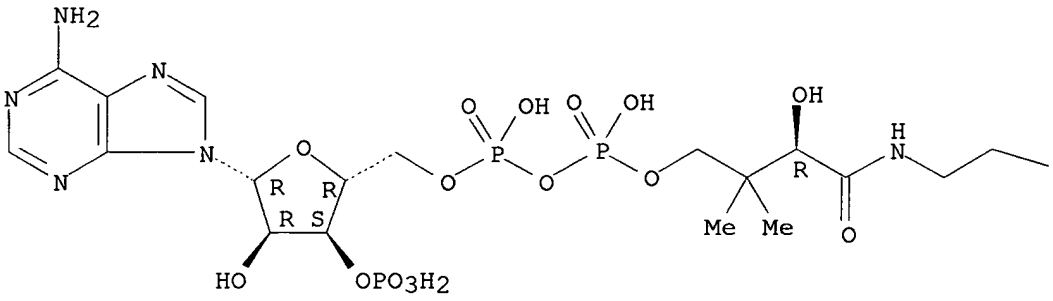
PAGE 1-B



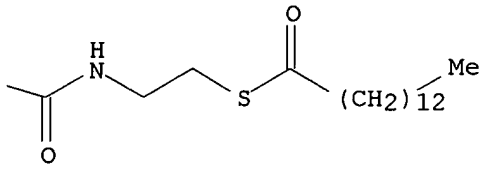
RN 3130-72-1 CAPLUS
CN Coenzyme A, S-tetradecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



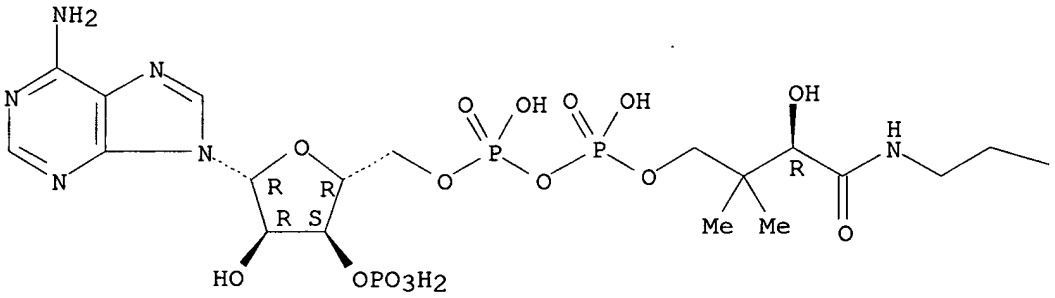
PAGE 1-B



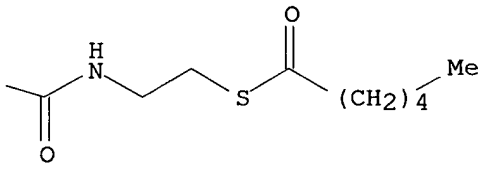
RN 5060-32-2 CAPLUS
CN Coenzyme A, S-hexanoate (7CI, 8CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



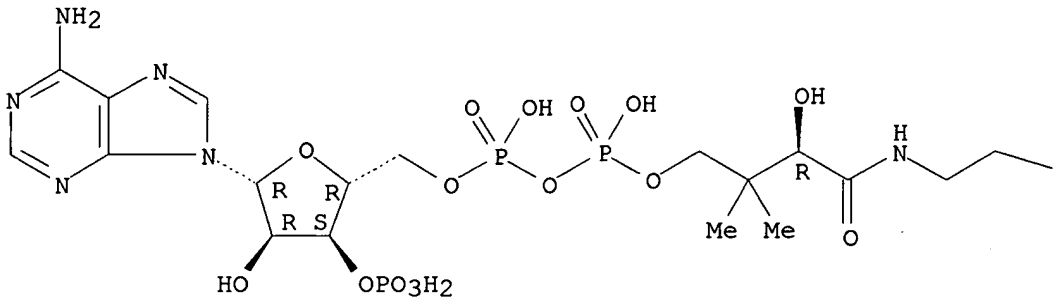
PAGE 1-B



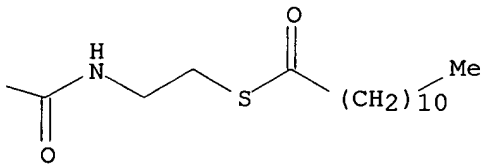
RN 6244-92-4 CAPLUS
CN Coenzyme A, S-dodecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



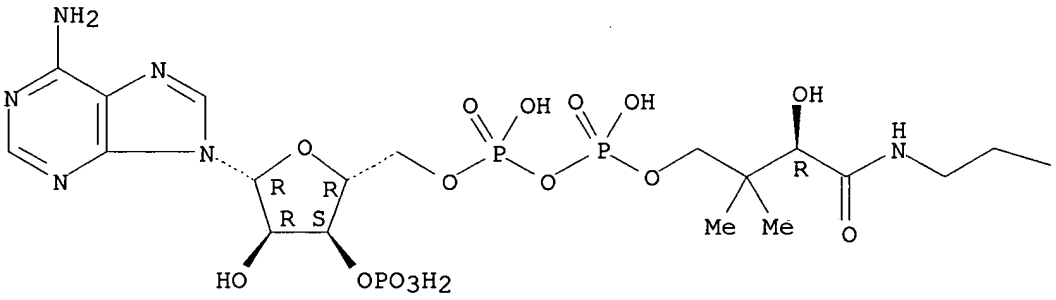
PAGE 1-B



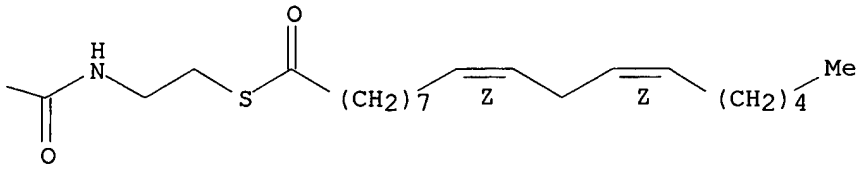
RN 6709-57-5 CAPLUS
CN Coenzyme A, S-(9Z,12Z)-9,12-octadecadienoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

PAGE 1-A



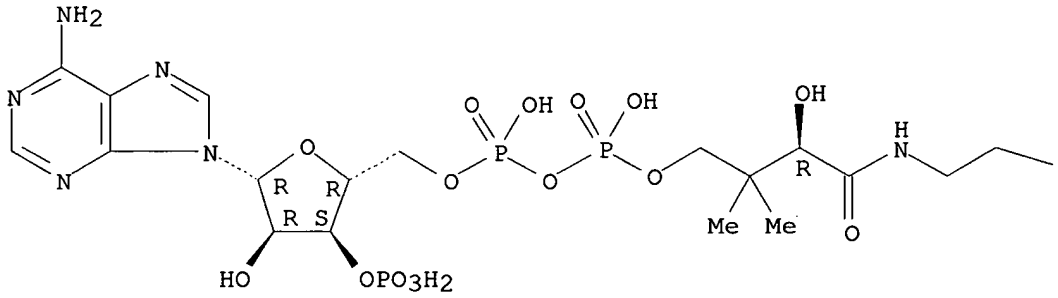
PAGE 1-B



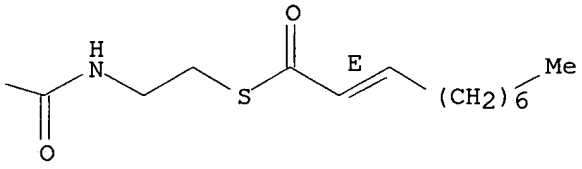
RN 10018-95-8 CAPLUS
CN Coenzyme A, S-(2E)-2-decenoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

PAGE 1-A



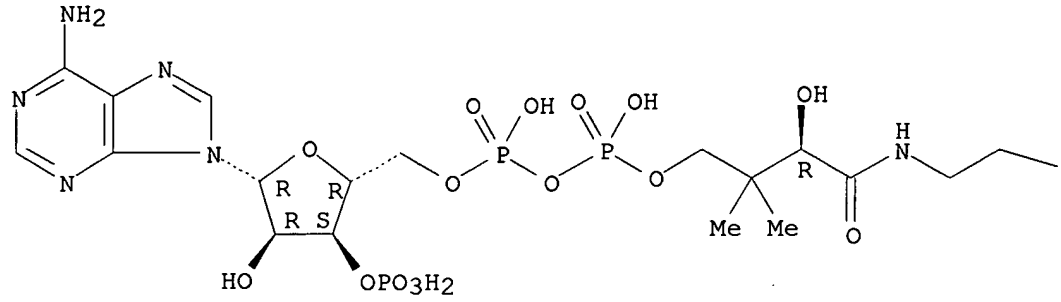
PAGE 1-B



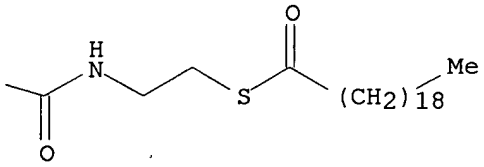
RN 15895-27-9 CAPLUS
CN Coenzyme A, S-eicosanoate (8CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



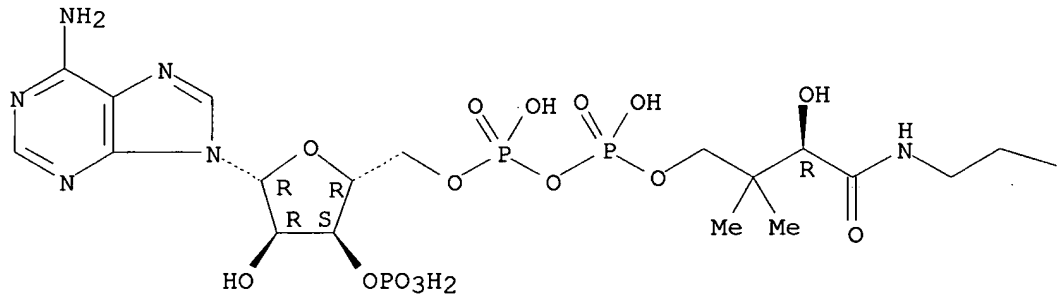
PAGE 1-B



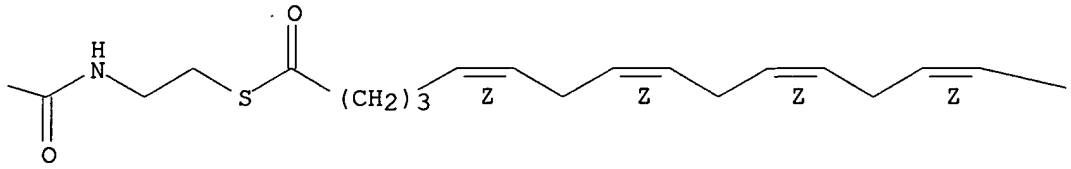
RN 17046-56-9 CAPLUS
CN Coenzyme A, S-(5Z,8Z,11Z,14Z)-5,8,11,14-eicosatetraenoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

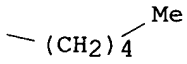
PAGE 1-A



PAGE 1-B



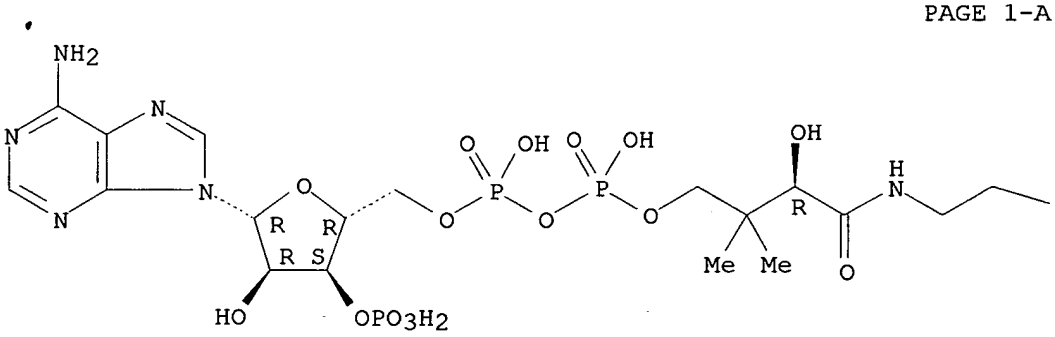
PAGE 1-C



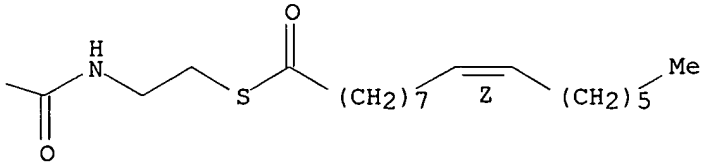
RN 18198-76-0 CAPLUS
CN Coenzyme A, S-(9Z)-9-hexadecenoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

PAGE 1-A



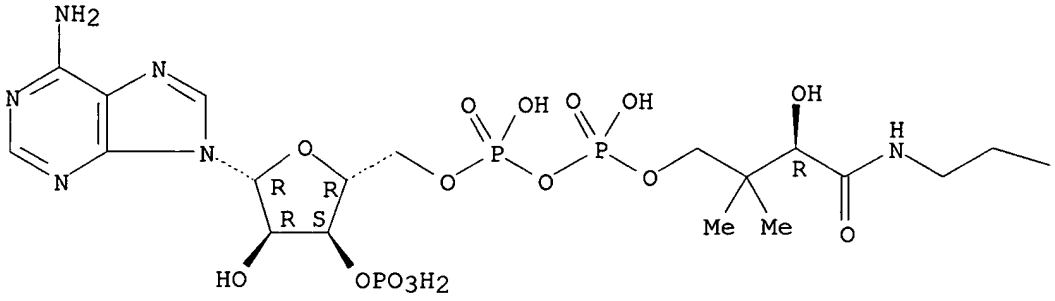
PAGE 1-B



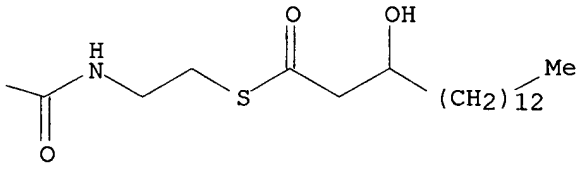
RN 35106-50-4 CAPLUS
CN Coenzyme A, S-(3-hydroxyhexadecanoate) (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



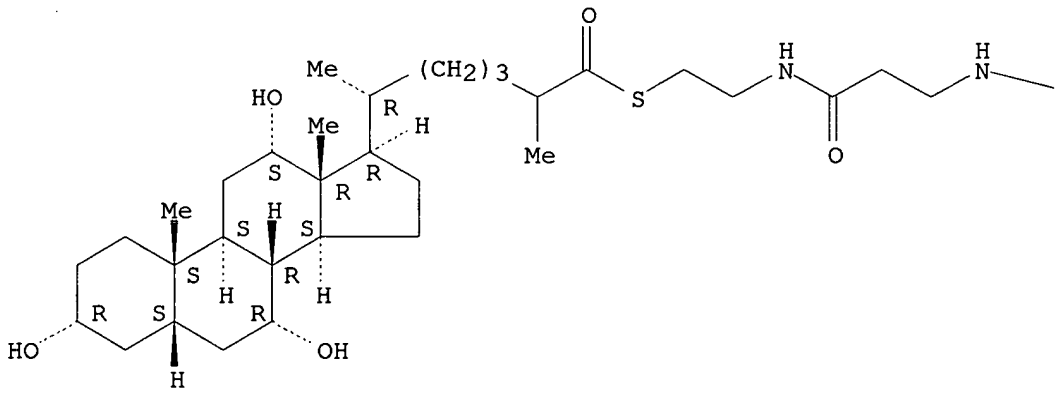
PAGE 1-B



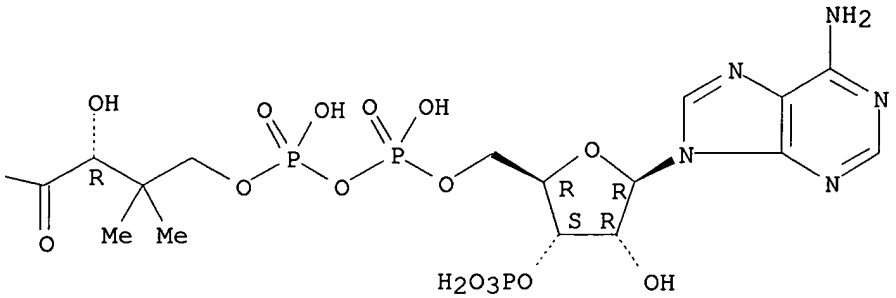
RN 57458-60-3 CAPLUS
CN Cholestane-26-thioic acid, 3,7,12-trihydroxy-, S-ester with coenzyme A, (3α,5β,7α,12α)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A

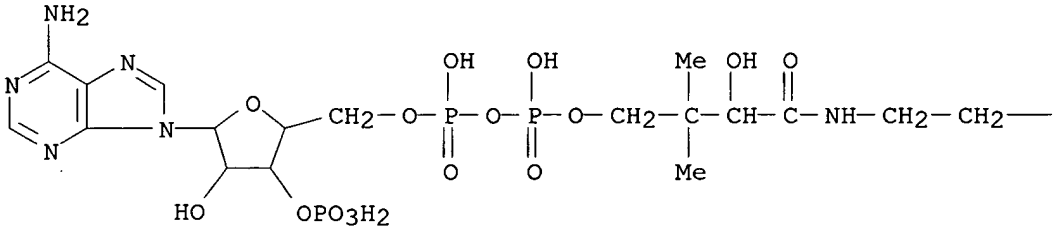


PAGE 1-B

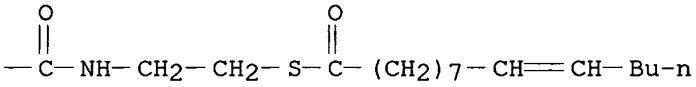


RN 87935-97-5 CAPLUS
CN Coenzyme A, S-(9Z)-9-tetradecenoate (9CI) (CA INDEX NAME)

PAGE 1-A



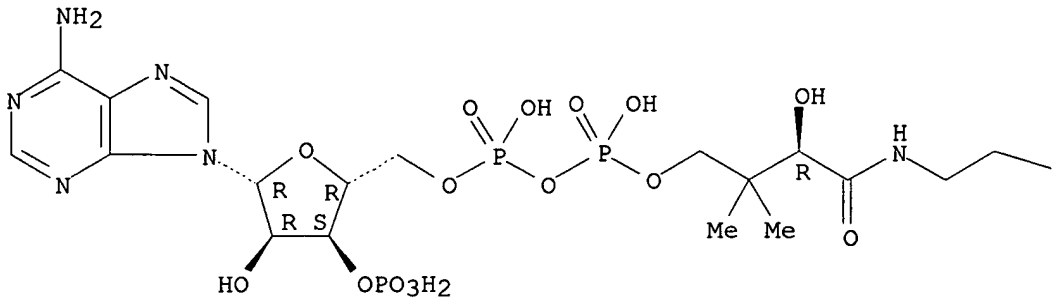
PAGE 1-B



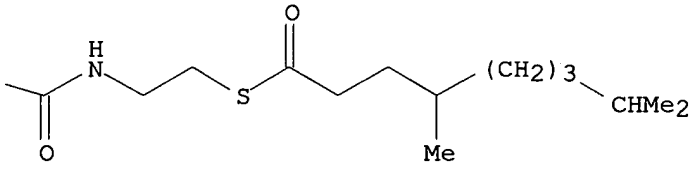
RN 204120-61-6 CAPLUS
CN Coenzyme A, S-(4,8-dimethylnonanoate) (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



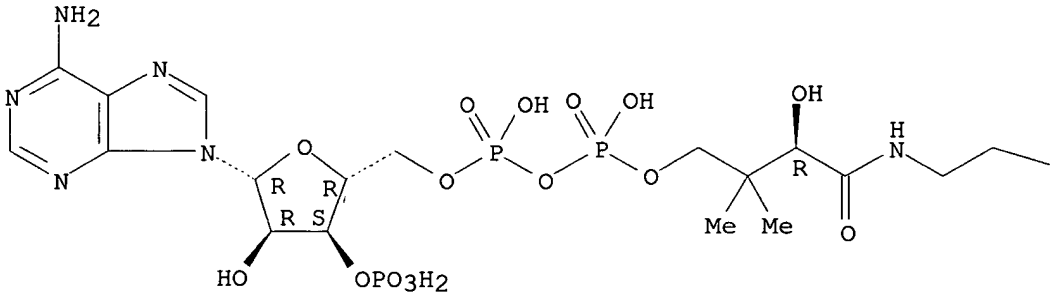
PAGE 1-B



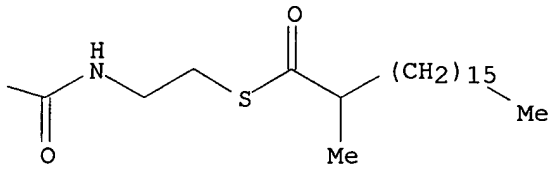
RN 409307-21-7 CAPLUS
CN Coenzyme A, S-(2-methyloctadecanoate) (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



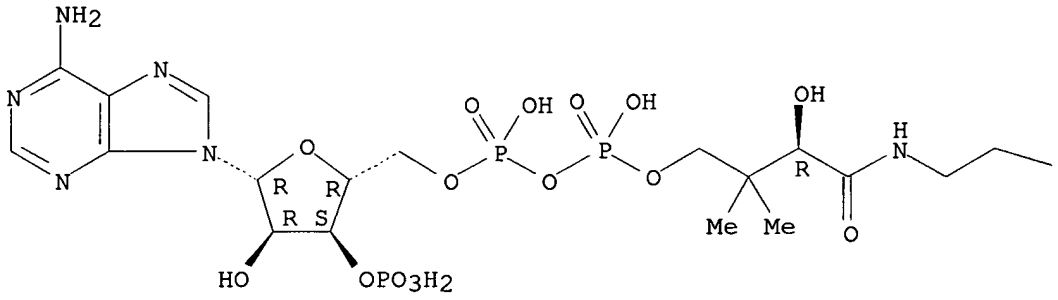
PAGE 1-B



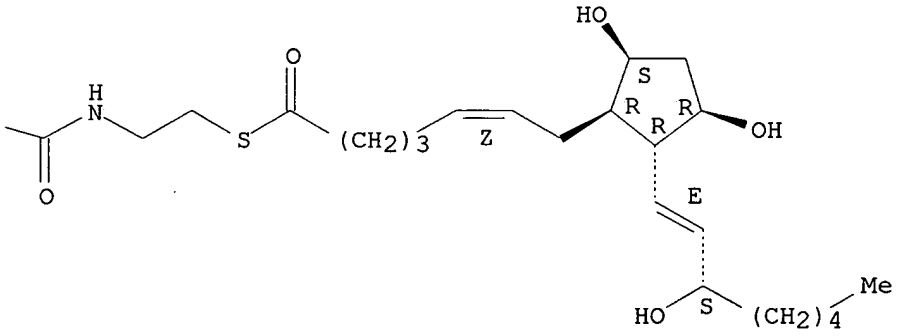
RN 434329-16-5 CAPLUS
CN Prosta-5,13-diene-1-thioic acid, 9,11,15-trihydroxy-, S-ester with coenzyme A, (5Z,9α,11α,13E,15S)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

PAGE 1-A



PAGE 1-B

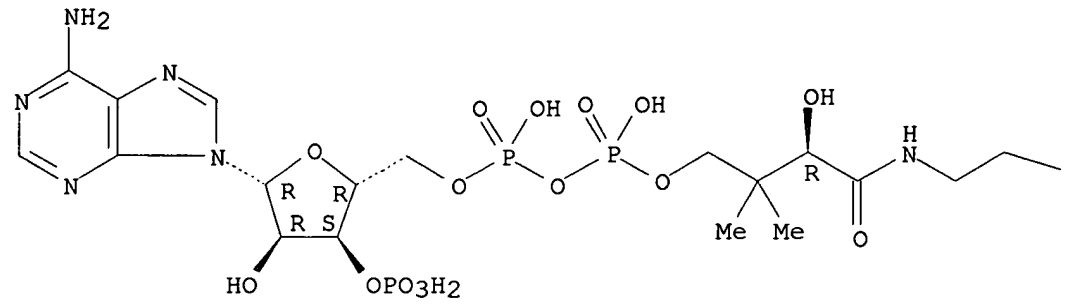


=> d his

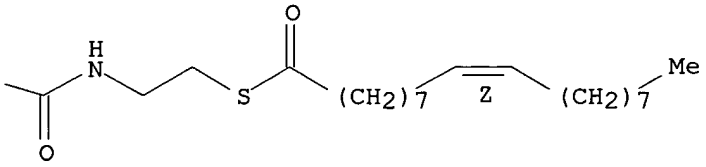
(FILE 'HOME' ENTERED AT 10:10:13 ON 04 OCT 2003)

```
     FILE 'REGISTRY' ENTERED AT 10:10:33 ON 04 OCT 2003
L1              STRUCTURE UPLOADED
L2           35 S L1
L3          643 S L1 FULL

     FILE 'CAPLUS' ENTERED AT 10:12:33 ON 04 OCT 2003
L4         2435 S L3
L5           47 S L4 AND (DIABETES OR OBESITY OR PPAR)
L6           52 S L4 AND P/DT
L7            1 S L5 AND L6
L8           46 S L5 NOT L7
L9           46 SORT L8 PY

=> s l3/prep and p/dt
          2435 L3
       3059166 PREP/RL
           154 L3/PREP
                 (L3 (L) PREP/RL)
       4191595 P/DT
L10          11 L3/PREP AND P/DT

=> sort py l10
SORT ENTIRE ANSWER SET? (Y)/N:.
PROCESSING COMPLETED FOR L10
L11          11 SORT L10 PY

=> d 1-11 cbib pi hitstr

L11  ANSWER 1 OF 11  CAPLUS  COPYRIGHT 2003 ACS on STN
1992:21058   Document No. 116:21058  Preparation of thioester and isostere
     analogs of oleoyl coenzyme A as well as 1,3-dioxane derivative as
     hypocholesterolemic agents.  Bloom, Jonathan D.; Dutia, Minu D. (American
     Cyanamid Co., USA).  U.S. US 5053426 A  19911001, 13 pp.  (English).
     CODEN: USXXAM.  APPLICATION: US 1990-501451 19900329.
     PATENT NO.        KIND  DATE           APPLICATION NO.   DATE
     ---------------   ----  --------       ---------------   --------
PI   US 5053426         A    19911001       US 1990-501451    19900329
     US 5173510         A    19921222       US 1991-725856    19910703
IT   1716-06-9DP, thioester and isostere analogs and dioxane derivs.
     RL: SPN (Synthetic preparation); PREP (Preparation)
        (preparation of, as hypocholesterolemics)
RN   1716-06-9  CAPLUS
CN   Coenzyme A, S-(9Z)-9-octadecenoate (9CI)  (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.
```

PAGE 1-A



PAGE 1-B



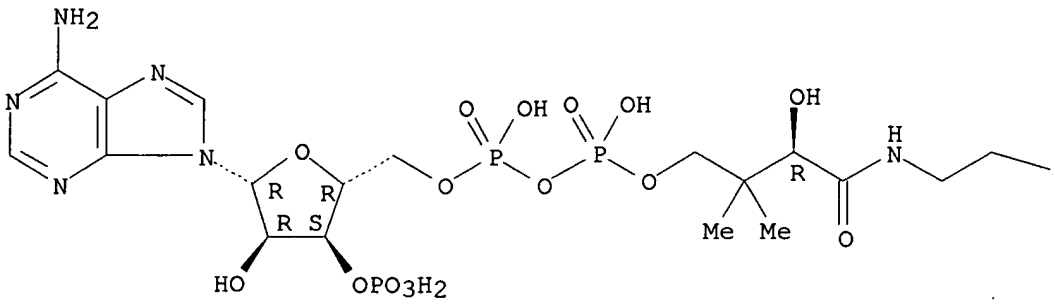
L11 ANSWER 2 OF 11 CAPLUS COPYRIGHT 2003 ACS on STN
1995:761488 Document No. 123:170077 Process for synthesizing fatty acid-acylated thiol transporter derivatives, particularly of acyl coenzyme A derivatives, and the acyl coenzyme A derivatives thus obtained.. Lellouche, Jean-Paul; Levannier, Karine; Mioskowski, Charles (Commissariat a l'Energie Atomique, Fr.). Eur. Pat. Appl. EP 618218 A1 19941005, 18 pp. DESIGNATED STATES: R: CH, DE, FR, GB, IT, LI. (French). CODEN: EPXXDW. APPLICATION: EP 1994-400675 19940329. PRIORITY: FR 1993-3744 19930331.

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	EP 618218	A1	19941005	EP 1994-400675	19940329
	R: CH, DE, FR, GB, IT, LI				
	FR 2703356	A1	19941007	FR 1993-3744	19930331
	FR 2703356	B1	19950512		
	US 5424415	A	19950613	US 1994-210300	19940318
	JP 06321973	A2	19941122	JP 1994-83603	19940331
	US 5519128	A	19960521	US 1995-392172	19950222

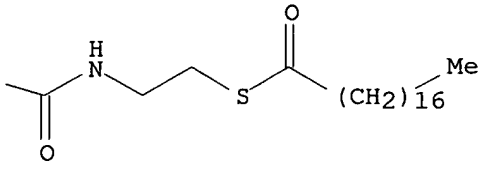
IT **362-66-3P**, Octadecanoyl coenzyme A **15895-27-9P**, Eicosanoyl coenzyme A **164666-53-9P**, 2-Fluoro-2-eicosenoyl coenzyme A **164666-55-1P**, 4-Fluoro-3-hydroxyeicosanoyl coenzyme A
RL: SPN (Synthetic preparation); **PREP (Preparation)**
(preparation of fatty acid-acylated thiol transporter (CoA) derivs.)
RN 362-66-3 CAPLUS
CN Coenzyme A, S-octadecanoate (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



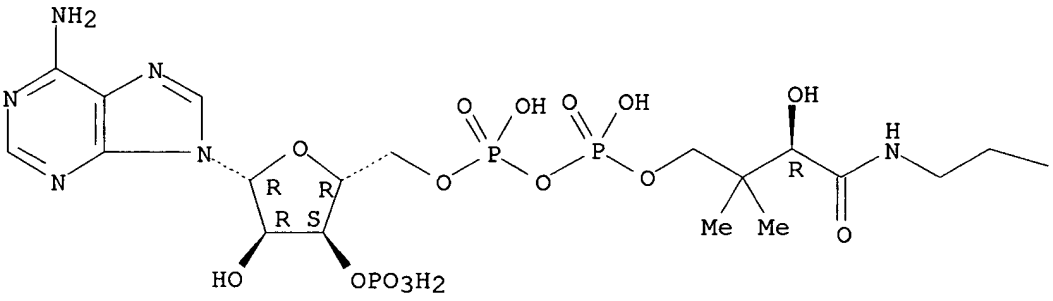
PAGE 1-B



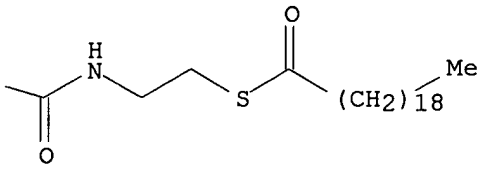
RN 15895-27-9 CAPLUS
CN Coenzyme A, S-eicosanoate (8CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



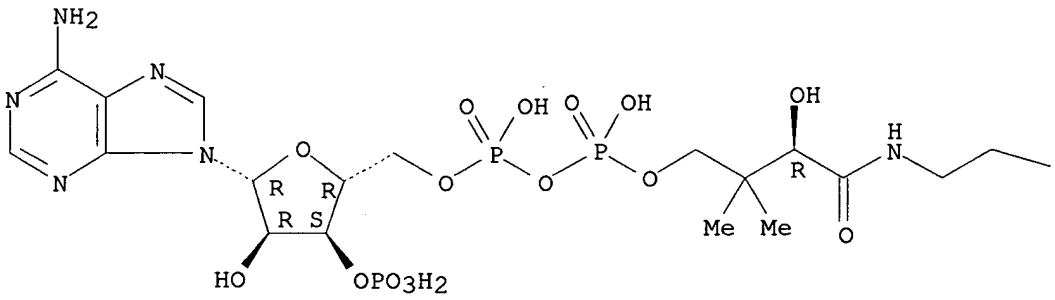
PAGE 1-B



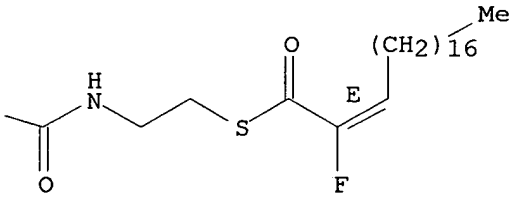
RN 164666-53-9 CAPLUS
CN Coenzyme A, S-(2-fluoro-2-eicosenoate), (E)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

PAGE 1-A



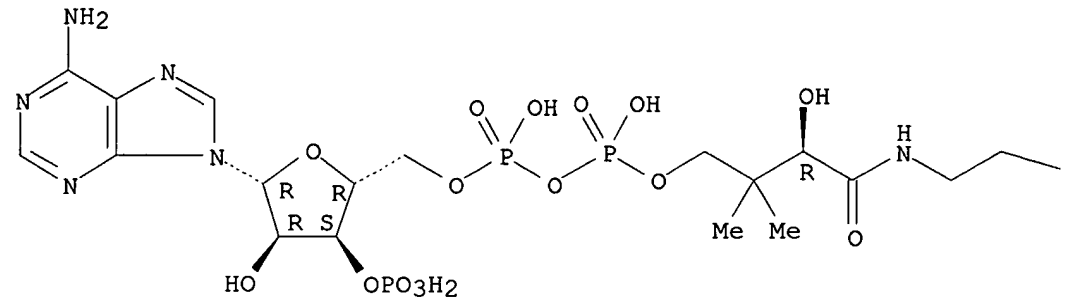
PAGE 1-B



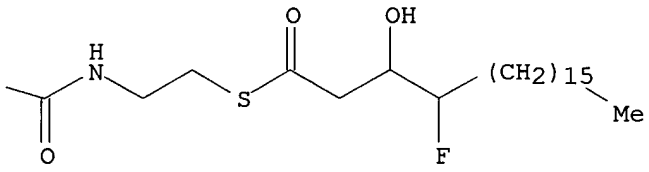
RN 164666-55-1 CAPLUS
CN Coenzyme A, S-(4-fluoro-3-hydroxyeicosanoate) (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B

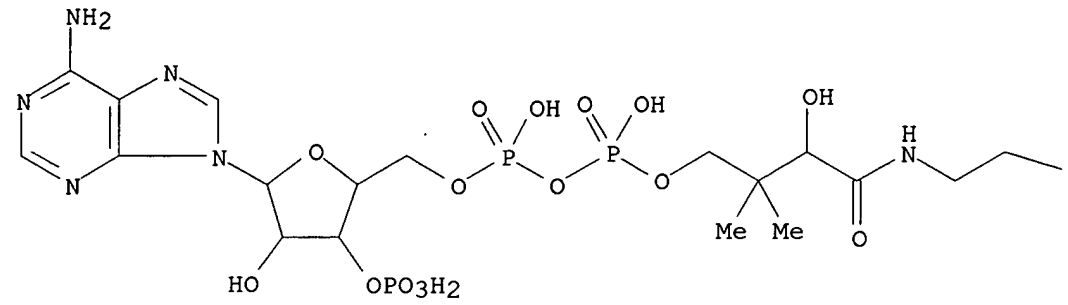


L11 ANSWER 3 OF 11 CAPLUS COPYRIGHT 2003 ACS on STN
1996:11000 Document No. 124:55805 Preparation of pyridylthioalkanoic acids and analogs as myristoylation-inhibiting antiviral agents. Ikeda, Shigeru; Hanya, Yoshitsugu; Shoji, Shozo (Torii Yakuhin Kk, Japan). Jpn. Kokai Tokkyo Koho JP 07215940 A2 19950815 Heisei, 7 pp. (Japanese).
CODEN: JKXXAF. APPLICATION: JP 1994-23541 19940127.

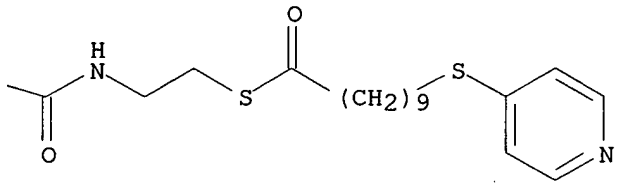
PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 07215940	A2	19950815	JP 1994-23541	19940127

IT **171967-49-0P**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); BIOL (Biological study); **PREP (Preparation)**
(preparation of pyridylthioalkanoic acids and analogs as myristoylation-inhibiting antiviral agents)
RN 171967-49-0 CAPLUS
CN Coenzyme A, S-[10-(4-pyridinylthio)decanoate] (9CI) (CA INDEX NAME)

PAGE 1-A



PAGE 1-B



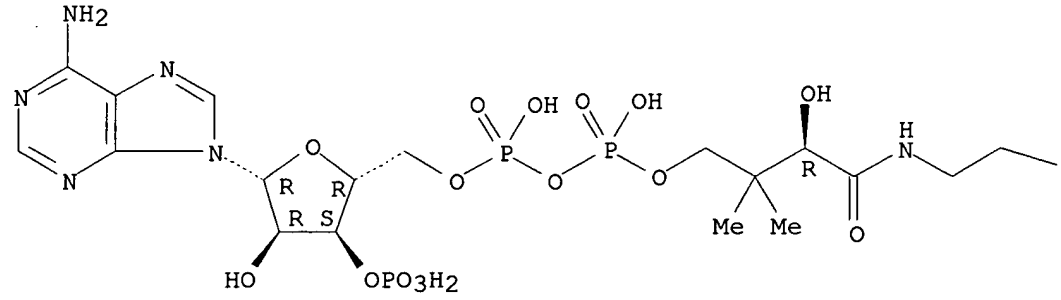
L11 ANSWER 4 OF 11 CAPLUS COPYRIGHT 2003 ACS on STN
1997:684502 Document No. 127:356534 An adipoyl-coenzyme A synthetase of Penicillium chrysogenum and its use in β-lactam biosynthesis. Bovenberg, Roelof Ary Lans; Hillenga, Derk Jans; Deen, Philippus Antonius; Pronk-Kraay Veld, Diana Esmeralda; Nieboer, Maarten (Gist-Brocades B.V., Neth.; Bovenberg, Roelof Ary Lans; Hillenga, Derk Jans; Deen, Philippus Antonius; Pronk-Kraay Veld, Diana Esmeralda; Nieboer, Maarten). PCT Int. Appl. WO 9738107 A1 19971016, 30 pp. DESIGNATED STATES: W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 1997-EP1711 19970404. PRIORITY: EP 1996-200902 19960404.

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9738107	A1	19971016	WO 1997-EP1711	19970404
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
	AU 9725088	A1	19971029	AU 1997-25088	19970404

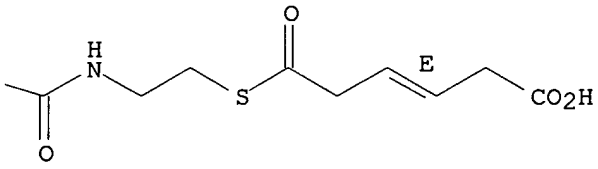
IT **198143-69-0P**
RL: BPN (Biosynthetic preparation); BIOL (Biological study); **PREP (Preparation)**
(enzymic preparation of; adipoyl-coenzyme synthetase of Penicillium chrysogenum and its use in β-lactam biosynthesis)
RN 198143-69-0 CAPLUS
CN Coenzyme A, S-[hydrogen (3E)-3-hexenedioate] (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

PAGE 1-A



PAGE 1-B



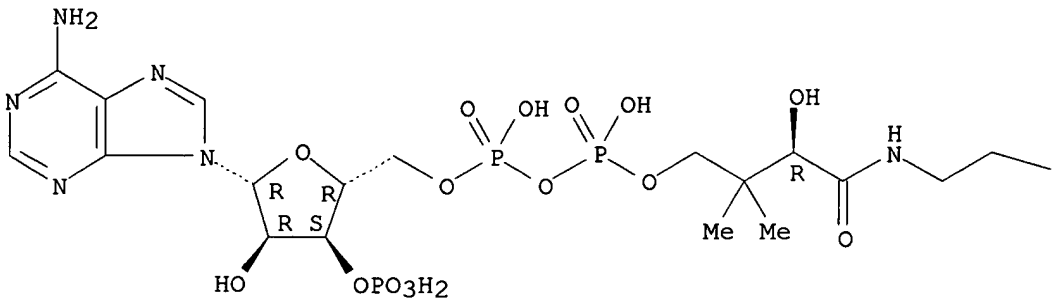
L11 ANSWER 5 OF 11 CAPLUS COPYRIGHT 2003 ACS on STN
1999:663215 Document No. 131:297109 Cloning of genes for enzymes associated with biosynthesis of d-biotin from Pseudomonas and use for d-biotin production. Yagasaki, Makoto; Nakamura, Noriko; Shibata, Susumu; Kino, Kuniki; Ikeda, Masato (Kyowa Hakko Kogyo Co., Ltd., Japan). Jpn. Kokai Tokkyo Koho JP 11285385 A2 19991019 Heisei, 19 pp. (Japanese). CODEN: JKXXAF. APPLICATION: JP 1998-89987 19980402.

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 11285385	A2	19991019	JP 1998-89987	19980402

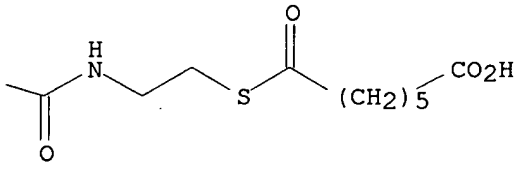
IT **18907-20-5P**, Pimelyl CoA
RL: BPN (Biosynthetic preparation); RCT (Reactant); BIOL (Biological study); **PREP (Preparation)**; RACT (Reactant or reagent)
(cloning of genes for enzymes associated with biosynthesis of d-biotin from Pseudomonas and use for d-biotin production)
RN 18907-20-5 CAPLUS
CN Coenzyme A, S-(hydrogen heptanedioate) (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



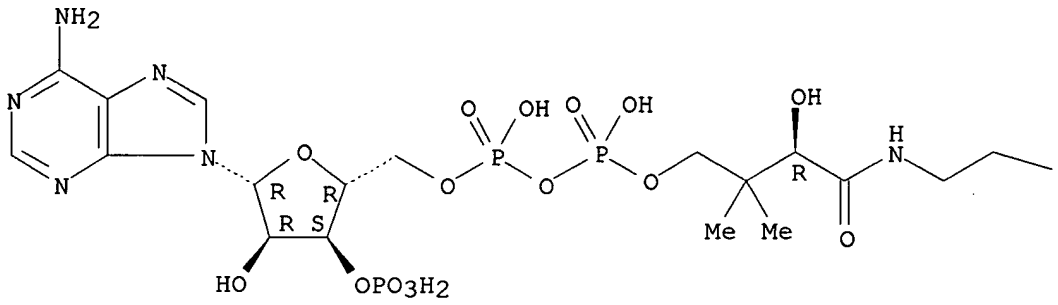
L11 ANSWER 6 OF 11 CAPLUS COPYRIGHT 2003 ACS on STN
1999:133267 Document No. 130:233991 Cloning of gene for pimelyl CoA synthetase from Brevundimonas diminuta. Hatakeyama, Kazuhisa; Kato, Yukie; Kobayashi, Mikio; Yukawa, Hideaki (Mitsubishi Chemical Industries Ltd., Japan). Jpn. Kokai Tokkyo Koho JP 11046763 A2 19990223 Heisei, 11 pp. (Japanese). CODEN: JKXXAF. APPLICATION: JP 1997-205791 19970731.

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	JP 11046763	A2	19990223	JP 1997-205791	19970731

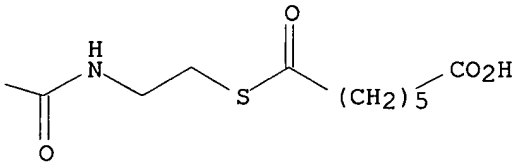
IT **18907-20-5P**, Pimelyl coenzyme A
RL: BPN (Biosynthetic preparation); BIOL (Biological study); **PREP (Preparation)**
(cloning of gene for pimelyl CoA synthetase from Brevundimonas diminuta for preparation of)
RN 18907-20-5 CAPLUS
CN Coenzyme A, S-(hydrogen heptanedioate) (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



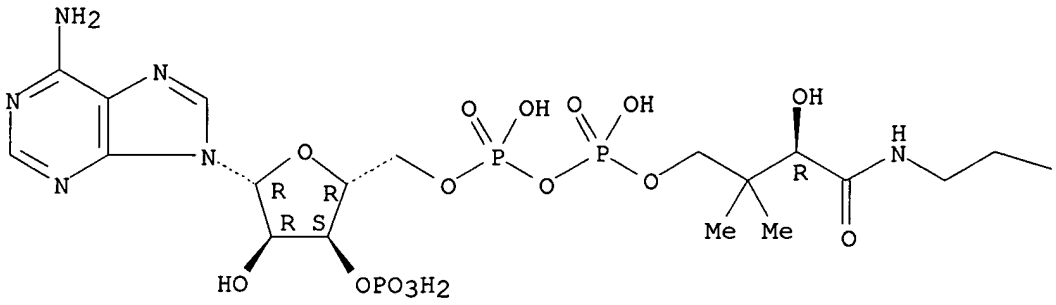
L11 ANSWER 7 OF 11 CAPLUS COPYRIGHT 2003 ACS on STN
2002:866703 Document No. 137:371428 Polyhydroxyalkanoate-covered pigment-containing ink and production method thereof. Nomoto, Tsuyoshi; Yano, Tetsuya; Kozaki, Shinya; Honma, Tsutomu (Canon Kabushiki Kaisha, Japan). Eur. Pat. Appl. EP 1256606 A2 20021113, 61 pp. DESIGNATED STATES: R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR. (English). CODEN: EPXXDW. APPLICATION: EP 2002-9670 20020429. PRIORITY: JP 2001-133550 20010427; JP 2001-210050 20010710.

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI EP 1256606	A2	20021113	EP 2002-9670	20020429
EP 1256606	A3	20030924		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
JP 2003012984	A2	20030115	JP 2001-210050	20010710

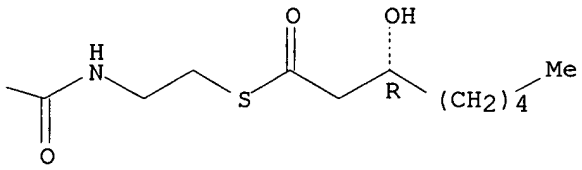
IT **117249-49-7P 473994-66-0P 474903-11-2P**
RL: IMF (Industrial manufacture); RCT (Reactant); **PREP (Preparation)**; RACT (Reactant or reagent)
(polyhydroxyalkanoate-covered pigment-containing ink and production method thereof)
RN 117249-49-7 CAPLUS
CN Coenzyme A, S-[(3R)-3-hydroxyoctanoate] (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



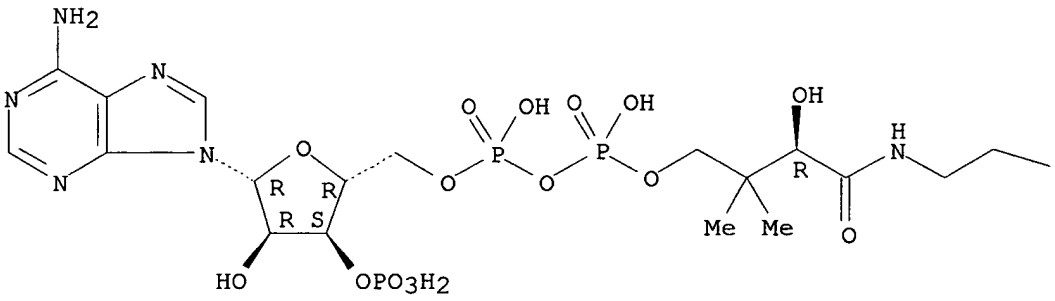
PAGE 1-B



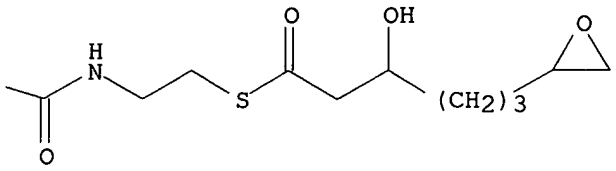
RN 473994-66-0 CAPLUS
CN Coenzyme A, S-(β-hydroxyoxiranehexanoate) (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



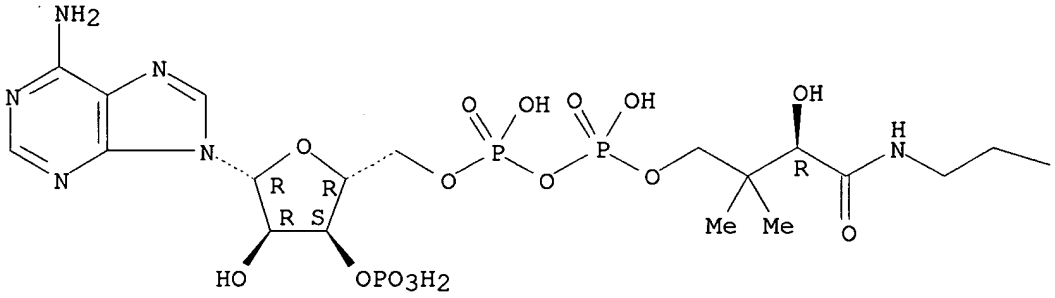
PAGE 1-B



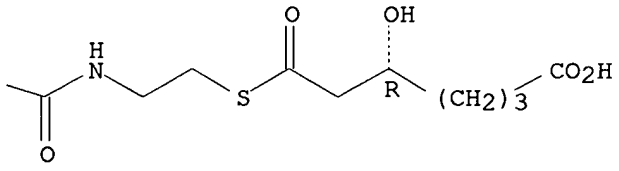
RN 474903-11-2 CAPLUS
CN Coenzyme A, S-[7-hydrogen (3R)-3-hydroxyheptanedioate] (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



L11 ANSWER 8 OF 11 CAPLUS COPYRIGHT 2003 ACS on STN
2002:847539 Document No. 137:354037 Microbial polyhydroxyalkanoate-based particles for electrophoretic display. Nomoto, Tsuyoshi; Yano, Tetsuya; Kozaki, Shinya; Honma, Tsutomu (Canon Kabushiki Kaisha, Japan). Eur. Pat. Appl. EP 1254930 A2 20021106, 61 pp. DESIGNATED STATES: R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR. (English). CODEN: EPXXDW. APPLICATION: EP 2002-9674 20020429. PRIORITY: JP 2001-131824 20010427; JP 2001-210060 20010710.

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI EP 1254930	A2	20021106	EP 2002-9674	20020429

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR

JP 2003015168 A2 20030115 JP 2001-210060 20010710

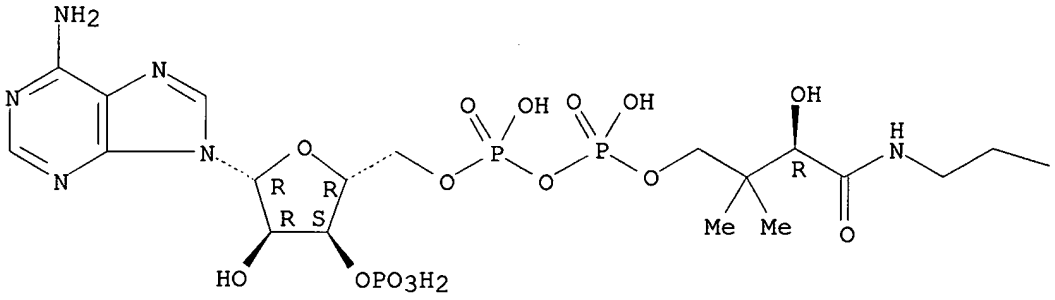
IT **117249-49-7P**
RL: IMF (Industrial manufacture); RCT (Reactant); **PREP (Preparation)**; RACT (Reactant or reagent)
(monomer; microbial polyhydroxyalkanoate-based particles for electrophoretic display)

RN 117249-49-7 CAPLUS

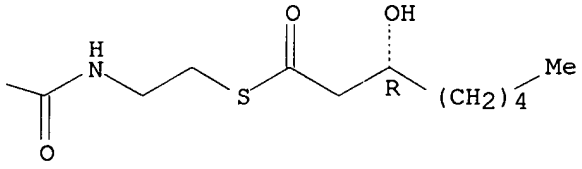
CN Coenzyme A, S-[(3R)-3-hydroxyoctanoate] (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



L11 ANSWER 9 OF 11 CAPLUS COPYRIGHT 2003 ACS on STN

2002:831899 Document No. 137:343851 Electrostatic charge image developing toner and image forming method. Yano, Tetsuya; Nomoto, Tsuyoshi; Kozaki, Shinya; Honma, Tsutomu (Canon Kabushiki Kaisha, Japan). Eur. Pat. Appl. EP 1253475 A2 20021030, 80 pp. DESIGNATED STATES: R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR. (English). CODEN: EPXXDW. APPLICATION: EP 2002-9673 20020429. PRIORITY: JP 2001-133728 20010427; JP 2001-210021 20010710.

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 1253475	A2	20021030	EP 2002-9673	20020429

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR

JP 2003015359 A2 20030117 JP 2001-210021 20010710

US 2003118931 A1 20030626 US 2002-133670 20020429

IT **117249-49-7DP**, reaction products with polyhydroxyalkanoates
RL: BCP (Biochemical process); PNU (Preparation, unclassified); TEM

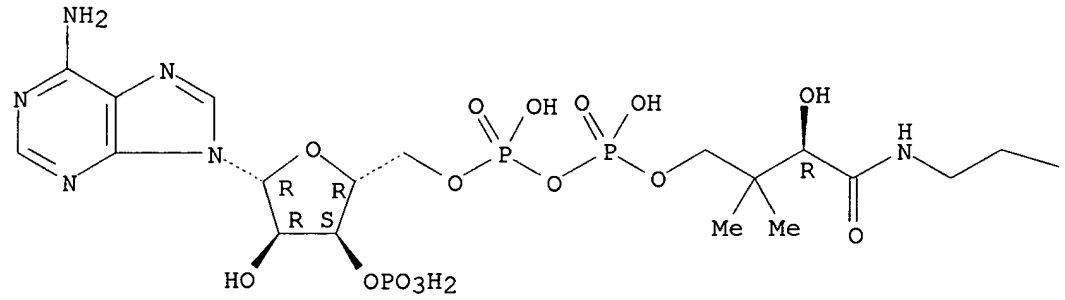
(Technical or engineered material use); BIOL (Biological study); **PREP (Preparation)**; PROC (Process); USES (Uses)
(electrostatic charge image developing toner comprising surface modified coloring agent)

RN 117249-49-7 CAPLUS

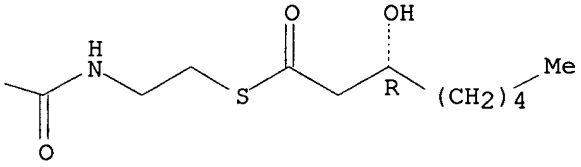
CN Coenzyme A, S-[(3R)-3-hydroxyoctanoate] (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



L11 ANSWER 10 OF 11 CAPLUS COPYRIGHT 2003 ACS on STN

2002:555622 Document No. 137:121608 Mutagenesis and crystal structure of polyketide synthases, and methods of altering the activity and substrate specificity of polyketide synthases. Noel, Joseph P.; Austin, Michael B.; Bowman, Marrianne E. (The Salk Institute for Biological Studies, USA). PCT Int. Appl. WO 2002057418 A2 20020725, 243 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2001-US48523 20011214. PRIORITY: US 2000-PV255811 20001215.

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002057418	A2	20020725	WO 2001-US48523	20011214
WO 2002057418	A3	20030821		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,

GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

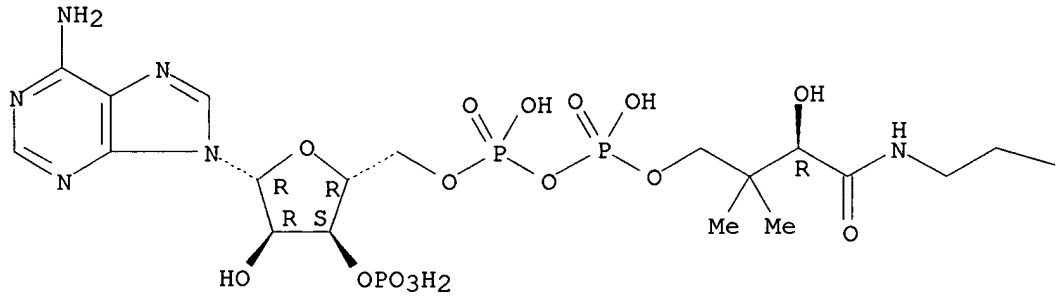
IT **5060-32-2DP**, Hexanoyl-CoA, complexes with chalcone synthase
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); **PREP (Preparation)**
(crystal structure; mutagenesis and crystal structure of polyketide synthases, and methods of altering activity and substrate specificity of polyketide synthases)

RN 5060-32-2 CAPLUS

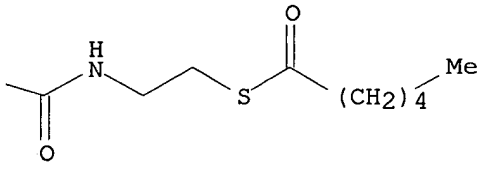
CN Coenzyme A, S-hexanoate (7CI, 8CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



L11 ANSWER 11 OF 11 CAPLUS COPYRIGHT 2003 ACS on STN

2003:40202 Document No. 138:108252 Immobilization of polyhydroxyalkanoate synthase on pigments and use in toner manufacture. Nomoto, Tsuyoshi; Yano, Tetsuya; Kozaki, Shinya; Honma, Tsutomu (Canon Kabushiki Kaisha, Japan). Eur. Pat. Appl. EP 1275728 A1 20030115, 277 pp. DESIGNATED STATES: R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK. (English). CODEN: EPXXDW. APPLICATION: EP 2002-15374 20020710. PRIORITY: JP 2001-210052 20010710; JP 2002-172978 20020613.

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

PI EP 1275728 A1 20030115 EP 2002-15374 20020710
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK

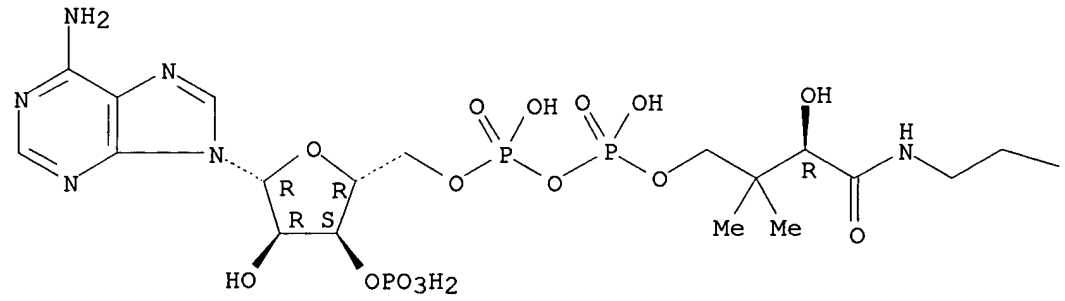
IT **117249-49-7P**
RL: BCP (Biochemical process); RCT (Reactant); SPN (Synthetic preparation); BIOL (Biological study); **PREP (Preparation)**; PROC (Process); RACT (Reactant or reagent)
(immobilization of polyhydroxyalkanoate synthase on pigments and use in toner manufacture)

RN 117249-49-7 CAPLUS

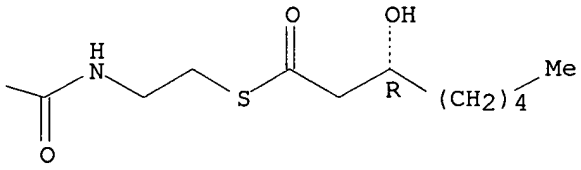
CN Coenzyme A, S-[(3R)-3-hydroxyoctanoate] (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



=> logoff
ALL L# QUERIES AND ANSWER SETS ARE DELETED AT LOGOFF
LOGOFF? (Y)/N/HOLD:.

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	234.29	383.45

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-26.04	-26.04

STN INTERNATIONAL LOGOFF AT 10:22:48 ON 04 OCT 2003